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VOL. I. No. 1 JANUARY, 1939

BRITISH HEART JOURNAL

FOR THE STUDY OF
CARDIOLOGY AND THE CIRCULATION

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APPOINTED BY
THE CARDIAC SOCIETY OF GREAT BRITAIN AND IRELAND

LONDON
BRITISH MEDICAL ASSOCIATION
TAVISTOCK SQUARE, W.C.1

PUBLISHED QUARTERLY SUBSCRIPTION 25/- PER ANNUM

CONTENTS

		PAGE
Foreword	By Sir Thomas Lewis	1
Some Disturbances of the Rhythm of the Heart	By John Cowan	3
Digitalis in Heart Failure with Normal Rhythm	By C. J. Gavey and John Parkinson	27
Chest Leads in Clinical Electrocardiography	By Paul Wood and A. Selzer	49
A New Sign of Left Ventricular Failure	By Paul Wood and A. Selzer	81
The Significance of Electrocardiograms showing a "Second Positive Wave of QRS" in Lead III . .	By A. A. Fitzgerald Peel	86
Some Notes on the Cardiac Club .	By John Cowan and Others	97

Præcordial Leads in Electrocardiography. Memorandum by the Cardiac Society and the American Heart Assoc. . . . 45

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FOREWORD

BY

SIR THOMAS LEWIS

The Cardiac Society of Great Britain and Ireland has decided that it is expedient for it to possess its own journal. The size of the society and the activity of many of its members render such a decision not only wise but inevitable.

There is no stronger tradition, concerning work upon the heart, than the British. Traced from the incomparable Harvey in the early part of the seventeenth century, through Richard Lower and Stephen Hales, the illustrious band of workers has been swollen by the great clinicians of the eighteenth and nineteenth centuries, Heberden, Parry, Withering, Corrigan, Hope, Stokes, Peacock, Ringer, Mackenzie, and by the great physiologists, Gaskell and Starling. All these names from the distant and recent past are linked with outstanding contributions to our knowledge of the heart and its maladies ; their work forms a legacy of great achievement. The members of the Cardiac Society of Great Britain and Ireland are the chief trustees of this legacy, and its weighty responsibility.

The success of the new journal will depend upon the quality and not primarily upon the quantity of the matter which it publishes. Originality of observation and of view will be the touchstone of quality. The work of editing is uncreative : an editor can publish only what is offered ; he will use most of what is offered. The journal's success is governed, therefore, from the source of the material. The best contributors will submit for publication only sound and original matter and thought. They will edit their own work ; will judge their own text impartially and will prune it ruthlessly, thinking of the standard of work accomplished and to be reported and not of personal interest ; they will prepare their manuscript in all particulars to save further labour ; they will facilitate the

reading by every device of conciseness, of simplicity, of clarity, and of accuracy, in composing text, figure explanation, and reference ; for every hour so spent by one who writes will save countless hours by the many who afterwards read. They will remember that the work of editing is a gratuitous and unenviable task, and they will bow loyally to editorial decision.

To express the hope that the new journal will come to possess many contributors of this kind is to give the fullest wishes for the journal's real and lasting success.

SOME DISTURBANCES OF THE RHYTHM OF THE HEART

BY

JOHN COWAN †

(*From the Royal Infirmary, Glasgow*)

I. INTRODUCTORY

The association of changes in the character of the pulse and serious cardiac symptoms was recognized many centuries ago, and carefully studied with all the means available at the time. But the significance of the arrhythmias has only been elucidated within the last few years, by the use of instrumental methods of analysis. In G. A. Gibson's text-book (1898), designed to be a mirror of the state of medical science and art at the close of the nineteenth century, only a couple of pages are devoted to the disturbances of the cardiac rhythm. James Mackenzie's book (1902) started an intensive study, and in his Morison Lectures, Gibson (1904) discussed paroxysmal tachycardia, extrasystole, and heart block. The study has continued, and our accumulated knowledge is now vastly greater than it was ten years ago, but there are still gaps on the clinical side. The following records are brought forward to augment the available data.

In the healthy individual each beat of the heart is occasioned by stimuli arising in the sino-auricular node, and spreading from it to the auricles and ventricles. The sinus pacemaker is under the control of the nervous system, and both the rhythm and the rate of the heart may be altered by nervous influences. "The heart responds to every emotion and sensation, and even thought, as well as to the demands created by muscular exertion or the organic processes such as digestion. . . . All states of the nervous system are reflected upon the circulation, each emotion being attended with its own reaction upon the heart and arteries, and even sensation producing recognizable results, so it is to be expected that diseases of the nervous system will be attended with special symptoms, manifested through the circulation, and by the pulse" (Broadbent, 1890). Stokes (1854) had previously referred to the influence of the nervous system upon the circulation, but he concluded that such disturbances of the pulse were more often related to functional disturbance rather than to organic disease of the nervous system, in contradistinction to disturb-

† Received June 15, 1938.

ances due to intrinsic cardiac causes, which were more often organic than functional.

Disturbances of the pulse may also arise from toxic causes, the activity of such poisons as digitalis or those of the infections ; though these doubtless act by their influence upon the nervous system or the heart itself.

In recent years attention has been largely focused upon the disturbances due to intrinsic cardiac causes ; those due to nervous and toxic causes have received little consideration. It is very important to recognize the nervous origin of an irregular pulse, for the mere suggestion of heart disease may cause incalculable mischief. We have all seen such cases. One came recently under my observation. I saw him first at the age of 13, when he gave a wonderful display of extrasystoles, clearly due to nervous causes. He was a big, healthy, growing schoolboy, who played all games with acceptance. He was advised to carry on ; but other advice was sought, and he was made an invalid. At the age of 27 he is an undersized, poorly developed, badly nourished hypochondriac. His heart still seems quite sound.

The present paper is mainly concerned with those disturbances of the cardiac rhythm which are due to causes *outwith* the heart.

The rhythm and the rate of the pulse in health are not absolutely regular. The rhythm, though apparently regular to the finger, is always irregular if accurately measured. During inspiration the rate is quickened and during expiration slowed. The rate of the pulse varies : fever may raise it to 120 per minute ; jaundice may lower it to 40 ; and comparable changes may occur from physical, psychical, or organic nervous influences. The variations are due to variations in the duration of diastole, the intrinsic mechanism of the heart continuing unchanged.

In health each beat of the heart is occasioned by stimuli arising in the sino-auricular node. Every part of the heart, however, possesses the inherent power of initiating stimuli, and the sino-auricular node only takes the lead because its rate of stimulus production (70–80 per minute) is more frequent than that elsewhere. The rate of the auriculo-ventricular node is 50–60 ; that of the ventricular muscle 20–30.

The heart may, at times, serve two masters. In many people stimuli occasionally arise elsewhere than in the sino-auricular node and cause a contraction : an extrasystole. If it concerns the ventricles alone, the normal sino-auricular rhythm of the auricles need not be disturbed, but an auricular extrasystole disturbs the sino-auricular rhythm for the moment.

If the rate set by the sino-auricular node becomes less than that set by the auriculo-ventricular node, the latter may take charge and cause a ventricular contraction. This may occur if the activity of the sino-auricular node is depressed so that its rate of stimulus production becomes less frequent than that of the auriculo-ventricular node. This can be caused, in experiment, by cooling the sino-auricular node ; by pressure upon the carotid sinus or the eyeballs ; by heavy doses of digitalis or quinidine. Cushny (1925) showed that in digitalis poisoning in animals, the rate of the auricular contractions

became less frequent and the irritability of the ventricles exaggerated, so that spontaneous contractions of the ventricles ensued, and, becoming more frequent, eventually established an " idio-ventricular rhythm," with a rate that might be equal to or more frequent than that of the auricles. The mechanism was clearly nervous, for if the vagus inhibition was put out of action by atropine the normal rhythm was restored.

The rate of stimulus production of the sino-auricular node may also become less frequent than that of the auriculo-ventricular node if the latter becomes hyper-irritable. This can be accomplished, in experiment, by warming the auriculo-ventricular node ; by the exhibition of atropine throwing the vagus nerve out of action ; by stimulation of the right vagus and left sympathetic nerves. In some cases more than one mechanism may be simultaneously in action.

As a rule, when the auriculo-ventricular node takes charge, the stimulus passes in a retrograde fashion to the auricles and causes an auricular contraction. In the case of an isolated auriculo-ventricular nodal extrasysfole, this may not occur if the nodal stimulus reaches the auricle at a time when its muscle is refractory ; but if the stimuli are repeated the auricular rhythm is generally set by the auriculo-ventricular node (Nodal rhythm). In exceptional cases this retrograde conduction is disturbed, and the auricular contractions arise in response to stimuli originating in the sino-auricular node, while the ventricular contractions are in response to stimuli from the auriculo-ventricular node (Dissociation). But, curiously, in some cases where retrograde conduction is impeded the forward conduction of stimuli is not altogether inhibited, and at rare intervals the ventricles may respond to a sino-auricular stimulus flowing through the auricular muscle (Capture : dissociation with interference).

The majority of these cases are associated with an infrequent sinus rate and occur during the administration of digitalis ; but dissociation may occur apart from such drugging, the result of purely nervous causes.

The most frequent cause of dissociation is, of course, heart-block, due to damage to the auriculo-ventricular bundle, interrupting the passage of the stimuli from auricle to ventricle. The auricles continue to contract at the sinus rate, whatever it may be, while the ventricles contract infrequently, 20–30 times a minute. The pulse rate in heart-block is less frequent than in dissociation, for the ventricular contractions are due to stimuli arising in the muscle of the ventricle, and not, as in dissociation, in the auriculo-ventricular node. The interruption of the pathway between auricles and ventricles in full heart-block prevents the sino-auricular node from regaining control of the ventricular contractions even on occasion.

Heart-block is generally due to organic lesions in the tissues concerned, but may be of functional origin. It has been produced experimentally by digitalis and quinidine intoxication ; by vagal stimulation ; by asphyxia ; in anaphylactic reactions. It has been produced clinically by digitalis, and by pressure upon the carotid sinus or the eyeballs. If of functional origin it can

be removed by atropine; but if due to organic lesions atropine is inactive, and the ventricles pursue their slow rhythm uninfluenced by exertion, atropine, or vagal stimulation. An exception to this statement has been reported by Fleming and Stevenson (1928). A child, aged 3½ years, with full heart-block and congenital heart disease, showed an increase in the auricular and the ventricular rates on the exhibition of atropine, although the block persisted unchanged.

The rate and the rhythm of the ventricular contractions may thus be affected by variations in the " tone " of the sino-auricular and the auriculo-ventricular nodes ; and this may be due to functional or organic disturbances, depressing or exalting their particular functions. If the rate of the stimulus production in the two nodes is approximately similar, trifling alterations in rate of either may alter the rhythm for the time. Sinus irregularities, sino-auricular block, nodal rhythm, dissociation, extrasystoles may thus all occur, in quick succession or on separate occasions, in the same patient, as will appear in the following clinical records.

Disturbances of the rhythm of the heart may be due to intrinsic disorders of the heart or to causes arising outwith the organ. The prognosis and the treatment of the two groups vary according to the cause of the disturbance.

II. ARRHYTHMIAS OF THE SINO-AURICULAR NODE

Sinus Arrhythmia.—Sinus irregularity of the pulse occurs most notably in children and young adults. The rate and the rhythm of the pulse are unstable. The intervals between the beats are variable, and diastole may be considerably prolonged (Fig. 1). The irregularity is often associated with the phases of

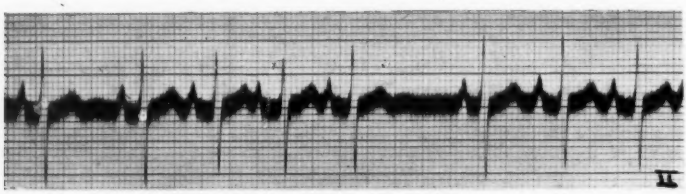

FIG. 1.—Sinus arrhythmia. The only abnormal feature is the varying length of diastole. Lead II.

respiration, the rate quickening with inspiration and slowing with expiration. It is most distinct when the heart is beating slowly, and is lessened or abolished if the rate is increased, as, for instance, by exertion, or the administration of atropine. It can generally be influenced by voluntary changes in the respiratory rhythm, frequent, infrequent, or irregular breathing. The patients show active reflexes and are often highly strung.

It is rarely difficult to recognize sinus arrhythmia. Its relation to the respiratory phases and its disappearance with an increased pulse rate make its nature clear. The electrocardiogram shows variations in the duration of diastole and normal complexes.

Sinus arrhythmia is a physiological occurrence, and does not require any alteration of the usual habits of the individual, or any treatment.

Sino-Auricular Block : Type A, with Ventricular Standstill.—As already mentioned, the rhythm of the sino-auricular node is not perfectly regular. On occasion a sinus stimulus may default and the pulse misses a beat, neither auricle or ventricle contracting : sino-auricular block. The duration of the diastole is usually double that of the normal rhythm, but it may be somewhat shorter. Sometimes several beats in series may fail, and the standstill may last for several seconds.

Case A. Fig. 2 shows sino-auricular block. An active old man, aged 71, when stooping to pick up some papers, tumbled down unconscious. He recovered in a

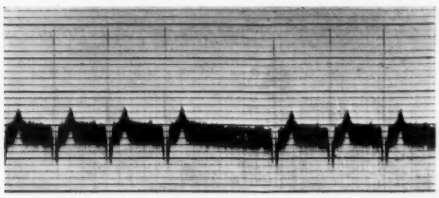

FIG. 2.—Sino-auricular block ; type A. Case A. A single contraction of the heart fails. Lead I.

few moments, but his face was bruised and he stayed at home for a day or two before resuming work. A similar attack occurred a year later. Two months afterwards, in August, he was rather short of breath upon exertion, and he had a third syncope when out walking, but was able to return home. His pulse was now found, for the first time, to be infrequent and irregular. In September he looked healthy and active, but his lips were somewhat blue. The left heart was slightly enlarged ; the first sound was impure ; the second aortic sound emphatic. His pulse was regular, save for an occasional extrasystole, and infrequent, 30–40 per minute. The electrocardiogram showed dissociation with interference (see Fig. 14, p. 16).

The pulse rate remained unaltered for a fortnight, and then gradually rose, reaching normal figures (60–72) before the end of a month. In December he looked well, but did not feel inclined for much work. His pulse at first was regular, save for an occasional extrasystole, and numbered about 66. But while electrocardiograms were being taken pauses, of which he was unconscious, occurred between the cardiac contractions (Fig. 2). The standstills varied in duration. Sometimes a single beat failed, sometimes several. One standstill lasted for 3·3 seconds.

His subsequent life was short. On January 5 he felt bilious, and stayed in bed. He became drowsy on the 7th, the stupor deepened, and he died in coma on January 10. No paralysis was detected, but the pupils and the reflexes became unequal and the breathing of Cheyne-Stokes type. The pulse rate was unaltered, but the cardiac contractions ceased for a second or two at intervals. Eight pauses were recorded in a tracing during 123·5 seconds, and while *a. c.* and *v.* were clear with each beat in the jugulo-carotid curve the line was straight during the pauses.

The striking feature of his illness was the almost complete absence of symptoms of cardiac disability. The breathlessness experienced in August disappeared after two or three weeks and did not recur. There were never any signs of congestive failure. The end came with symptoms of cerebral character, and the sino-auricular block seemed due to a similar cause.

Case B. The patient was a lonely old bachelor, aged 54, who was seen for some trivial digestive disturbance. His organs seemed sound, but his pulse rate was infrequent, about 47 per minute, and irregular, a sinus irregularity with sino-auricular block (Fig. 3). His discomforts rapidly passed away. Two years later he complained

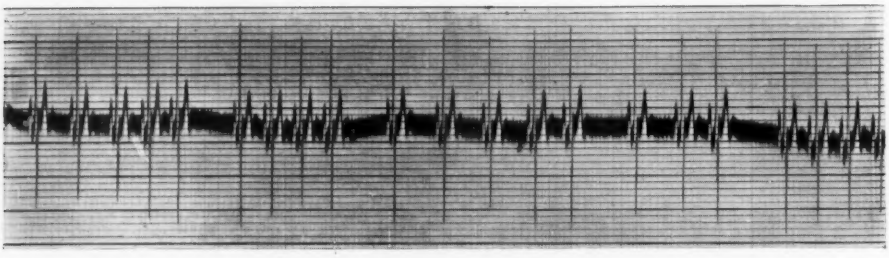

FIG. 3.—Sinus arrhythmia and sino-auricular block : type A. Case B. The record shows a series of progressively shortening auricular diastoles, leading up to long diastolic pauses. The disturbance of the auricular rhythm may be due to extreme sinus arrhythmia or to a sino-auricular block which displays features analogous to Wenckebach periods in auriculo-ventricular block. Lead II.

of his sense of taste ; his electrocardiogram showed an almost regular pulse of 43 per minute and normal complexes. Nine years later he is in good health. There was clearly a neurotic factor in all his complaints.

I can find few clinical records of cardiac standstill from sino-auricular block. Laslett's case (1908) is interesting, for the patient has now been under observation for nearly thirty years. A married woman, she enjoyed good health until the age of 36 (1904), when she became subject to syncopal attacks. In 1908 she was slightly anæmic and rather breathless on exertion. Some hæmic murmurs were audible on auscultation, but her organs otherwise seemed sound. The attacks recurred at intervals of three or four weeks, and were repeated for several days. The pulse, which usually was regular in rhythm and about 70 per minute, on these occasions became infrequent (30–40), as the heart often failed to contract, the result of sino-auricular block. If the standstill lasted for 4–5 seconds she lost consciousness, and if for longer the fingers might twitch. On one occasion she lost her speech for a week. Dr. Laslett courteously informs me (1937) that the attacks still recur as frequently as thirty years ago. She had a stroke two years ago, which has left her slightly aphasic and very decrepit, but she has never shown any signs of cardiac insufficiency. The rhythm of her heart is very sensitive to pressure upon the carotid sinus, and long pauses between the contractions are readily produced in this way.

Heard and Strauss (1918) reported the case of a woman, aged 36, with tonsillitis and a dry pleurisy. She had been liable to " faints " since the age of 16. These attacks commenced with discomfort in the precordial region, and she became short of breath and cyanosed. On several occasions she lost consciousness. Attacks lasted for 15–60 minutes, and she felt weak for a day or two afterwards. They had become more frequent and more severe, and she had been an invalid for a year. She was undersized and nervous. The

Wassermann reaction was positive. Her uterus was undeveloped, but the other organs seemed sound. The pulse was very unstable, the rate, usually 40–70, varying between 40 and 126 per minute. On one occasion it ran 18–25 for a short period. The electrocardiogram showed a sinus arrhythmia and sino-auricular block. When she was under an anæsthetic, for curetting, the rhythm changed to an auriculo-ventricular nodal rhythm with a pulse rate of 83–97. Subsequently the rhythm became normal but later reverted to the original sinus irregularity.

Barlow (1927) reported several cases of sino-auricular block. A man, aged 65, who suffered from chronic arthritis, had an attack of giddiness one morning three years before he came under observation, with pain in the shoulder and breathlessness. His blood pressure was 190/90 mm., his heart was enlarged, and a systolic murmur was audible at the aortic cartilage. Sino-auricular block persisted for several months, but he had few discomforts. Another man, aged 41, complained of breathlessness, faintness, and exhaustion of two years' duration. His blood pressure was 140/70 mm., his heart of normal size, his mitral valve incompetent. The Wassermann reaction was positive. The electrocardiogram showed sino-auricular block. His condition remained unaltered for the next two years ; and three years later he merely complained of lassitude although the block persisted.

In three of these cases syncopal attacks occurred on more than one occasion. The common causes of syncope are vaso-motor disturbances, and failure of the pulse from any cause, producing anæmia of the brain. If the failure is momentary the patient becomes pale ; if it lasts for 4–5 seconds consciousness is lost ; if for 10–12 seconds tonic or clonic convulsions supervene.

Attacks can be produced by pressure upon the carotid arteries. They occur in about one-third of the cases of heart-block most frequently at the period when the rhythm is changing from a partial to a complete block, and ventricular standstills occur from time to time. They are uncommon after full block has been established. I have recorded (1926) their occurrence in a case of paroxysmal nodal tachycardia, where the pulse failed during the paroxysms (Fig. 4).

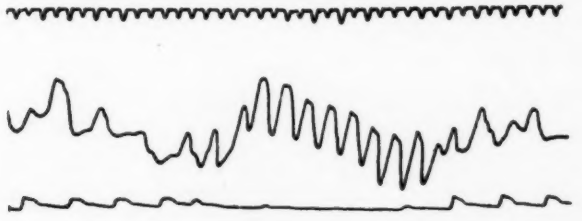

FIG. 4.—Polygraph tracing showing failure of the radial pulse during a short attack of nodal tachycardia. Case L, p. 19. P.R. 160.

Syncope may occur in sino-auricular block if the standstill is sufficiently

prolonged. The sequence was clearly proven in Laslett's case. The association of attacks of giddiness, faintness, and syncopes with periods of ventricular standstill in my case and that of Heard and Strauss suggest a similar origin ; but no attack occurred under observation.

Sino-Auricular Block : Type B.—When the sino-auricular stimulus is late in arriving and the auriculo-ventricular node supplies a stimulus for the ventricular contractions.

In this group of cases of sino-auricular block ventricular standstills do not persist for any length of time. On the failure of an auricular stimulus and the consequent prolongation of diastole, the inherent rhythmicity of the auriculo-ventricular node comes into action and evokes a ventricular beat (Fig. 5). As a rule the sinus node regains control of the ventricular contractions

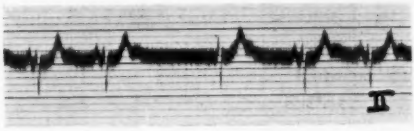

FIG. 5.—Sino-auricular block : type B. Case C. Following the failure of a sinus stimulus and undue prolongation of diastole the auriculo-ventricular node sets the ventricular rhythm for one beat. Lead II.

after but one failure, but occasionally the auriculo-ventricular nodal rhythm continues for several cycles (Fig. 6). In some cases the change of rhythm recurs repeatedly in an irregular fashion.

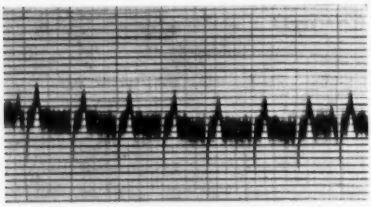

FIG. 6.—Sino-auricular block : type B. Case D. Following the failure of a sinus stimulus and undue prolongation of diastole the auriculo-ventricular node sets the ventricular rhythm for six beats. Lead II.

Case C. An active man, aged 57, had been liable to headaches, but otherwise had enjoyed good health until four years prior to his visit, when he became subject to indigestion and various muscular pains. He was thin and nervous, and looked older than his years. His organs seemed sound, but his pulse, numbering about 60 per minute, was very irregular. The electrocardiogram showed a fairly regular sino-auricular rhythm, with at times sino-auricular block : type B (Fig. 5).

His health improved though the irregularity persisted, and he continued at work for five years. At this time, after business worries, he had to enter a mental hospital, where he remained for four months. He was always eccentric in his habits and he had a family history of mental troubles. A year later he was seen again ; he looked stout and well, and only complained of slight breathlessness and discomfort in the chest upon exertion. His heart was larger than before and his blood pressure higher,

170/85 mm. Hg., but there was little evidence of cardiac mischief. His pulse was infrequent, about 38 per minute, and very irregular as the pacemaker " wandered." In some records a fairly regular sinus rhythm continued for several beats, and was suddenly succeeded by a series of ventricular complexes without any evidence of auricular activity. On two occasions an inverted P was visible between RS and T (Fig. 7). In Fig. 8, taken just after Fig. 7, a positive P immediately preceded QRS

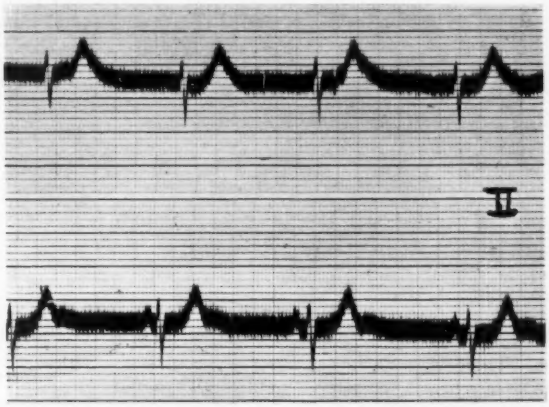

FIG. 7.—Sino-auricular block : type B. Case C. The upper record shows four beats of auriculo-ventricular nodal origin. An inverted P is visible between RS and T in the third complex. The lower record shows a return to sino-auricular control. Lead II.

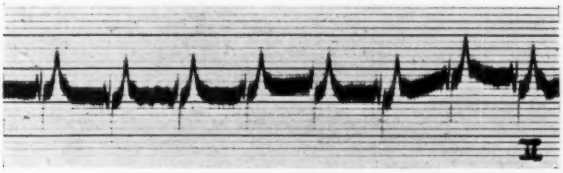

FIG. 8.—Dissociation. Case C. The sinus rhythm is infrequent and irregular. The second auricular diastole is prolonged, and the auriculo-ventricular node, impatient at the delay of the auricular stimulus, sets the ventricular rhythm for the next three beats, after which the sinus rhythm becomes re-established. Lead II.

merging into the upstroke of R. The interpretation is difficult on account of the varying rate of the auricular contractions. The inverted P waves are evidently due to retrograde auricular activation, and their close proximity to QRS seems to preclude the possibility of P deflexions being buried in the other ventricular complexes. Probably the auricles were at rest save on these occasions : a high-grade sino-auricular block. The sinus rhythm in Fig. 8 is infrequent and irregular. The second auricular diastole is prolonged so the auriculo-ventricular node, impatient at the delay of the auricular stimulus, sets the ventricular rhythm for the next three beats (dissociation), after which the sinus rhythm becomes re-established.

The records are interesting as they show the persistence of comparable cardiac irregularities over a period of at least six years, without any appreciable cardiac difficulties even during his severe mental illness.

Case D. A man, aged 58, was seen after an attack of influenza. He felt uncomfortable when walking, but he would not admit of any dyspnœa, palpitation, or œdema. He had become liable to attacks of giddiness, and had lost consciousness on several occasions. His heart was normal in size. Systolic murmurs were audible in all areas. His arteries were degenerate and his blood pressure was 155/110 mm. His pulse numbered about 66 per minute and was irregular—a fairly regular sino-auricular rhythm, with at times sino-auricular block, type B (Fig. 6).

After resting for a couple of months his health improved, though the sino-auricular block persisted. He returned to work, but after a year retired from business. Three years later anginous symptoms arose, and a year later he had a slight stroke. His heart was now enlarged, the mitral valve incompetent, and his blood pressure lower, 140/90 mm. His pulse was fairly regular and numbered about 66. The electrocardiogram showed a normal rhythm, but evidence of myocardial damage. He died a year later in an anginous attack.

In this patient sino-auricular block was present at a time when, although his heart was unsound, cardiac symptoms were in abeyance ; and had ceased, at a later date, when cardiac symptoms were clamant.

Case E. A well-to-do plumber came under observation in 1932, at the age of 61. He had contracted syphilis at 57. He had had a cough for many years, and was short of breath upon exertion, and liable to attacks of dyspnœa, sometimes without obvious cause. There was a chronic broncho-pneumonia at the left base. With care he regained fair health, leading a quiet life. In 1936, when he was seen after an attack of tonsillitis, merely complaining of shortness of breath upon exertion, the basal consolidation appeared to be larger than before.

In 1932 his heart seemed normal in size. The first sounds were rather weak and distant, and the second sound was doubled. His pulse numbered about 67 and was irregular ; there was a fairly regular sino-auricular rhythm, with at times sino-auricular block, type B (Fig. 9). In 1936 his heart seemed unaltered, but the doubling of the

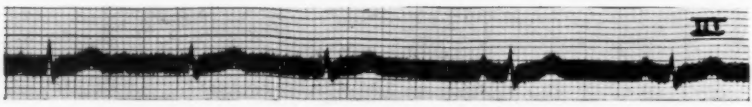

FIG. 9.—Sino-auricular block : type B. Case E : 1932. The first beat is of sinus origin and is followed by two beats in response to auriculo-ventricular nodal stimuli, succeeded by two beats of sinus origin. Lead III.

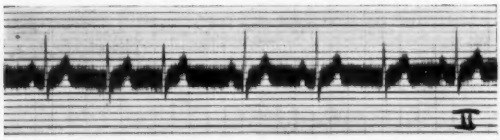

FIG. 10.—Sino-auricular block : type B. Case E : 1936. The second, third, and sixth beats are in response to auriculo-ventricular nodal stimuli. Lead II.

second sound had ceased. The pulse, 60–70 per minute, was again irregular. The electrocardiogram showed an intermittent sino-auricular block as before (Fig. 10). Both sets of records showed sinus irregularity and auriculo-ventricular nodal irregularity, the diastolic periods preceding the nodal beats being of very variable duration.

Case F. A man, aged 32, came under observation on account of breathlessness

and palpitation upon exertion. He had a well-marked mitral stenosis, of rheumatic origin. His pulse, numbering about 90 per minute, was irregular. The electrocardiogram showed slight sinus irregularity, and at times sino-auricular block, type B (Fig. 11). He remained in fair health for the next five years, save for occasional attacks of cardiac failure associated with bronchitis. He then had a hemiplegic seizure, and died shortly afterwards.

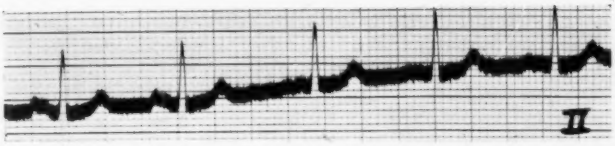

FIG. 11.—Sino-auricular block : type B. Case F. The last three beats are in response to auriculo-ventricular nodel stimuli. Lead II.

The following record is interesting as the changes in rhythm were associated with a cardiac infarct, the only instance in my experience.

Case G. A man, aged 63, had led a wandering life in his early days, been careless in his habits, and contracted many infections. For a year he had been liable to palpitation and giddiness upon exertion. A fortnight before his visit he had had a severe attack, and had been in bed for a few days.

He was a well-built man but obese. He did not look seriously ill, and a minimal amount of œdema on the shins was the only sign of cardiac insufficiency. His heart was large, and double murmurs were audible over the sternum. His blood pressure was 200/65 mm., and his arteries showed gross degenerative changes. It seemed clear that he had had an infarct. With care his health improved, and a year later he had few discomforts if he lived quietly. He had had several syncopal attacks, of short duration. His heart was larger than before, his blood pressure 245/70 mm., and the albuminuria considerable. He refused to rest ; and a few months later, when in church, he had another infarct, and died two days later.

Electrocardiograms were taken on three occasions. The first (1928) showed an irregular sinus bradycardia (Fig. 12), with a pulse rate of about 30 per minute. In the upper record the rhythm is apparently normal, while the lower shows dissociation with interference, the aberrant complex of the captured beat denoting impaired conduction in the bundle branch on account of the prematurity of the contraction. The second record, a year later, shows a variable sino-auricular block, the auricular rhythm being very irregular and infrequent, and the long pauses allowing the lower centre to capture, or supplement, the ventricular rhythm (Fig. 13). The last record. a fortnight later, showed an almost regular sinus rhythm, with a pulse rate of about 40. The P–R interval measured 0·18–0·20 in. (Fig. 13). The irregular sinus bradycardia may have been due to impairment of the blood supply to the sino-auricular node. Barker and Kinsella (1924) reported a case of sino-auricular block in a dog that had an acute streptococcic infection ; the almost normal sino-auricular node was completely surrounded by an inflammatory mass.

Cutts (1937) has recorded several cases in which the pacemaker " wandered " from the sino-auricular to the auriculo-ventricular node and back again, in an irregular fashion. The cause of the change of rhythm was evidently varied. One patient, aged 37, complained of general weakness and a tremor of the head for six years. Her pulse was known to have been infrequent for many years. At the time of observation it numbered about 48 per minute, and was irregular.

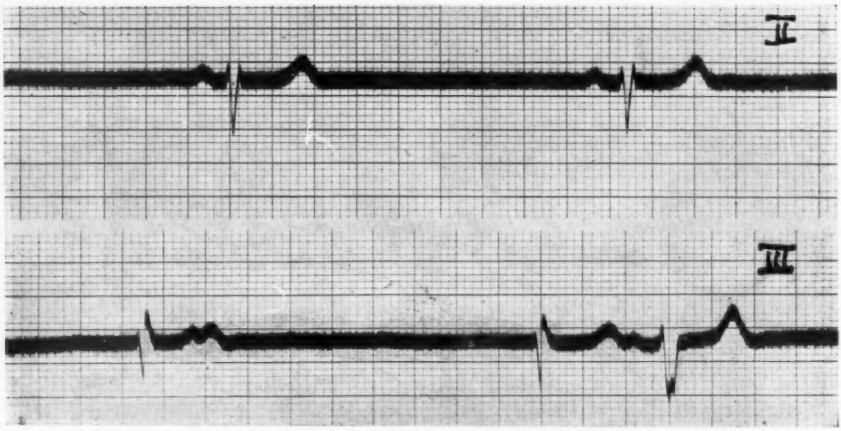

FIG. 12.—Dissociation with interference. Case G. The upper record shows an apparently normal sinus rhythm, with an abnormally slow rate, about 30 per minute. The lower record shows dissociation with capture of the third ventricular systole by the preceding auricular stimulus. The abnormal ventricular complex indicates impaired conduction in the bundle branch. Leads II and III.

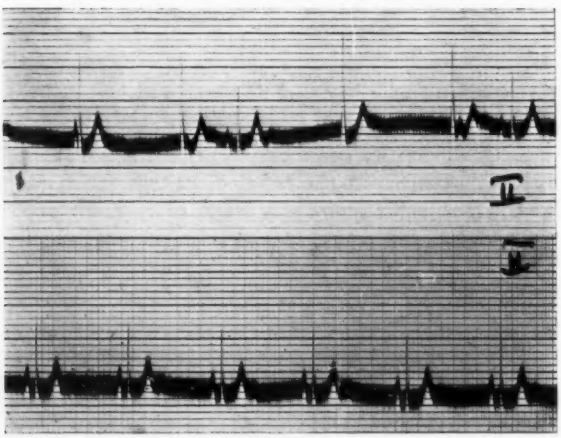

FIG. 13.—Sino-auricular block : type B. Case G. The upper record shows that the auriculo-ventricular node sets the ventricular rhythm whenever diastole is unduly prolonged. The lower record shows a return to normal sinus rhythm. Lead II.

Her heart seemed to be sound. She made a satisfactory recovery and was well six months later. There was a well-marked sinus irregularity, and when diastole was prolonged the auriculo-ventricular node took charge, and, as in some beats an inverted P was present between R and T, evidently originated these auricular contractions.

Another patient, aged 27, was suffering from rheumatic fever, and was fevered at the time of observation ; she had aortic and mitral valvular disease. The pulse numbered about 80, and was irregular. There was little difference in the rate set by the two nodes, and at times the auriculo-ventricular node took charge of the ventricular contractions. As the Ps were positive, when present, there was evidently a retrograde block. The data suggest an enhanced irritability of the auriculo-ventricular node as the cause of the altered rhythm. The patient's health improved, and she was sent home for further rest.

Some other reported cases appear to be examples of sino-auricular block, although the proof is not complete. Lewis (1925) refers to an athlete whose regular pulse of 36 when at rest abruptly rose to double that rate upon moderate exertion, and to a case reported by Neuburger and Edinger, where syncope frequently followed defæcation ; an aneurysm of the basilar artery, exerting pressure upon the medullary centres when the blood pressure rose during the act, seemed the probable cause of the attacks: and to Gerhardt's case, where similar attacks were associated with a tumour involving the left vagus nerve. Osler (1909) described a patient who suffered from attacks of syncope with an infrequent pulse of 10–12 per minute, associated with tubercular disease of the first and second cervical vertebræ. The case of de Zarday (1936) is definite ; in a woman, aged 42, suffering from a tumour in the posterior fossa of the skull, the pulse showed intermissions after each sixth systole, due to sino-auricular block. The intraspinal pressure was high, and a normal rhythm ensued for several hours after lumbar puncture. She died during an attempt at encephalography, but a post-mortem examination was not secured.

Conclusions

Sino-auricular block is not necessarily accompanied by cardiac symptoms. Four cases came under observation on account of symptoms other than cardiac ; nine cases on account of cardiac symptoms. The heart may be apparently sound (six cases) ; or may show signs of organic heart disease (seven cases).

Sino-auricular block can only be recognized by polygraphic or electrocardiographic examination. It may be suspected as the cause of recurrent syncopes in patients who show, between attacks, gross sinus irregularity.

The presence of sino-auricular block does not affect the cardiac prognosis or treatment in the individual case ; which must be based upon the general rather than the cardiac picture.

III. DISSOCIATION

Fig. 14 shows an example of dissociation with interference, from the old man (case A) reported on p. 7. His pulse then was fairly regular save for

occasional extrasystoles, the auricular rate 32·7 per minute, and the ventricular rate 32·8. At first the P precede waves QRS with a narrowing interval. Later

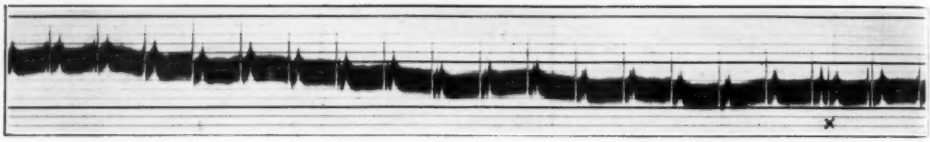

FIG. 14.—Dissociation with interference. Case A. See text. The ventricular beat 18 (x) is due to capture by the preceding auricular stimulus. Lead I.

they become lost in R and then emerge between R and T. Finally P fuses with T and again becomes invisible. As this auricular stimulus reached the ventricle at a time when it was excitable, it was followed by a ventricular contraction (x) : (capture). With care his pulse rate rose to normal figures, and 3 months later numbered 66 and was almost regular, P preceding QRS by about 0·20 in. On this occasion sino-auricular block was recorded (Fig. 2) ; it continued until his death a month later.

Case H. This patient has been under observation, at intervals, for many years. He was born in 1865. A well-built, muscular man, he engaged in athletic pursuits in early life, but permitted himself to become stout and flabby at an early age. He has had many adventures ; infections of varied character ; and has consistently " done himself well." In 1895 G. A. Gibson sent him to bed for cardiac symptoms, accompanied by œdema, which had followed athletic feats on the hill. In 1912 he became subject to attacks of giddiness, probably related to chronic otitic media, and had become unable to sustain continued exertion. " A lion before lunch ; a mouse after it." He made similar complaints in 1916 and 1921, and became very nervous. In 1925 he had two syncopes, following long drinks after shooting all day. He was now short of breath upon exertion. In 1930 he had to cease shooting as he tired easily. In 1936 he was leading a quiet life, and was very well.

His heart has always been normal in size, and his blood pressure has never been high ; his arteries are soft. In 1926 QRS was splintered and T3 inverted, but the Ts were positive after 1928. His pulse has been slow—51–57–64—and often irregular from extrasystoles ; in 1926 it showed functional dissociation without interference (Fig. 15). A similar irregularity was noted in 1930, but the rhythm was normal in 1912, 1916, 1921, and 1928.

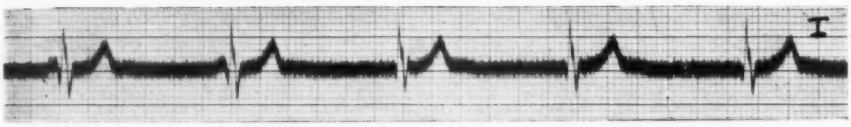

FIG. 15.—Dissociation. Case H. The P–R intervals of the first two beats are shortening, and the succeeding Ps are lost in QRS. Lead I.

Case I. This patient came under observation at the age of 53, on account of pain in an elbow following trauma. He suffered from chronic bronchitis and was liable to asthma. His heart seemed normal to physical examination, but the pulse was slightly irregular and an electrocardiogram showed well marked R–T deviation in leads II and III, and also slight sinus arrhythmia and auriculo-ventricular dissocia-

tion (Fig. 16). Three days later the sinus irregularity still persisted, but the rhythm was normal and the R–T deviation was less marked. Nine years later he is in fair health and at work. He has never shown any symptoms suggestive of cardiac insufficiency.

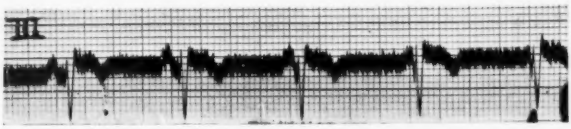

FIG. 16.—Dissociation. Case I. The P–R intervals of the first three beats are steadily shortening. The fourth P merges into R, and the fifth is lost in the ventricular complex. Lead III.

Another example of auriculo-ventricular dissociation was shown in Fig. 12, obtained from case G. White (1916) reported the case of a woman, aged 24, whose heart appeared to be sound. When convalescing after removal of the tonsils four days previously, her pulse was noticed to be slightly irregular and she complained of palpitation. The electrocardiogram showed dissociation, the auricles beating 67–85 times per minute and the ventricles 96. Next day the rhythm was normal, but dissociation was readily produced by pressure upon either carotid sinus, which slowed the auricular rate from 80 to 68, and thus permitted the escape of the auriculo-ventricular node. She was up and about at the time of the observation.

Hewlett (1923) reported the case of a man, aged 75, seen for shortness of breath, œdema of the feet, and pain in the chest upon exertion. His heart was enlarged, with a diastolic murmur sometimes audible at the apex, and a raised blood pressure, 175/110 mm. At intervals over a period of months dissociation was present ; generally the ventricular contractions (84, 88, 84, 75) were more frequent than the auricular (73, 71, 71, 72), but on one occasion the auricular rate was 75 and the ventricular rate 66. The pulse rate varied between 66 and 96. Atropine increased the ventricular rate and pressure over the carotid sinus slowed it. Large doses of digitalis slowed the auricular rate, but did not affect that of the ventricles.

Enescu and Vacareanu (1934) reported two cases of dissociation. One was suffering from rheumatic fever ; some sinus arrhythmia was present, with a pulse rate of 56. On the fourth day dissociation was recorded, there being but little difference between the auricular and the ventricular rates. The rhythm had returned to normal three days later. The second man, aged 32, complained of attacks of vertigo, and his pulse numbered about 68. There was usually sinus arrhythmia, and a P–R interval of 0·22 in. On one occasion dissociation was present, with a pulse rate of 61. The general picture suggested myocardial mischief. Neither of these patients had taken any digitalis.

Ritchie (1935) reported two cases of dissociation. One, a woman aged 44, was admitted into hospital on account of congestive failure, due to a diffuse myocardial fibrosis. After some improvement, the pulse rate falling from 90 to 60–63, she soon relapsed and digitalis was exhibited. The rhythm was

normal, but five days later interference dissociation was present, the auricular rate being 60·7 and the ventricular rate 73·1 per minute. She died two days later. His second case, also a woman aged 44, was suffering from congestive failure, with mitral stenosis. She had been taking digitalis for some months prior to admission. Dissociation was present, the auricular rates being 42, 45, 57 and the ventricular rates 75, 74, 87. It continued until her death eleven days later.

Conclusions

The conditions which were associated with the occurrence of dissociation in these ten cases were varied. In one it accompanied an infarct ; and in seven cases there was evidence of myocardial mischief. In two the heart was apparently sound. Dissociation is often associated with the administration of digitalis, and accompanied it in several of the cases mentioned above ; but in four cases no digitalis had been given.

There are no special symptoms associated with dissociation. In four of these patients symptoms of cardiac insufficiency were present, and two complained of cerebral symptoms. One was suffering from rheumatic fever, two merely complained of irregularity of the heart, and one made no complaint referable to the heart.

Dissociation can only be recognized by electrocardiograms. Its occurrence does not affect the outlook from the cardiac standpoint. It does not call for any specific treatment, but its frequent association with the administration of digitalis suggests that the dose, if not altogether discontinued, should be of moderate amount.

IV. THE NODAL RHYTHMS

The various forms of auriculo-ventricular nodal rhythm may be conveniently divided into three clinical groups :

(1) those with paroxysms of trachycardia ;

(2) those with a persistent frequent pulse rate ;

(3) those with the pulse rate unaltered, or less frequent than normal.

(1) *Paroxysmal Nodal Tachycardia.*—This group is the most common form of paroxysmal tachycardia (146 of 325 cases). It occurs at all ages, and need not occasion any distress save palpitation, unless, from long continuance of the tachycardia or pre-existing cardiac disease, the muscle of the heart becomes exhausted. The clinical story is varied.

Case J. A healthy boy of 16 had had attacks of palpitation at irregular intervals as long as he could remember. They lasted for variable periods, sometimes for several days. During the attacks the pulse ran about 220–240 per minute. Between attacks he was fit and well. His organs seemed sound.

Case K. An active country gentleman found, at the age of 69, that he was becoming subject to attacks of palpitation and shortness of breath upon exertion, steadily increasing in frequency and severity. During the attacks the pulse numbered about 136 per minute. His blood pressure, heart, and blood vessels seemed in reason-

able condition for his age. The electrocardiogram showed that P was hidden in R. After a few months hepatic colic ensued, and a gall-stone was removed successfully. The attacks then lessened in severity, and he became able to resume his former field activities. Ten years later he is leading a quiet life and in reasonable health. The attacks are now infrequent and of short duration.

If, however, the heart is already weak the outlook is serious.

Case L. A done old man, aged 77, was admitted into hospital complaining of attacks of giddiness, and of faints in some of which he had fallen to the ground. He was emaciated, childish, and restless, and steadily deteriorated both mentally and physically during his residence.

Many attacks were witnessed. The striking feature was failure of the pulse. If the failure was of short duration, a few seconds, he became restless and sometimes ceased to breathe ; if for 10 seconds he became very pale and lost consciousness. On several occasions when the pulse failed for a longer period the muscles twitched, and less often generalized convulsions ensued.

His heart was normal in size and site. The sounds were closed but short and distant. All his arteries were tortuous and thickened. His blood pressure varied : 115–155/60–95 mm. The cardiac rhythm was very unstable. His pulse might be regular even for days, but was often irregular from extrasystoles, which might be isolated events in a long series of regular beats, or, more often, bigeminal, trigeminal, or wholly irregular in incidence. When the radial pulse failed the cervical veins at once became greatly distended, pulsating vigorously at about double their usual rate (160 per minute). Polygraph tracings showed large venous waves and lessened arterial volume (Fig. 4). The electrocardiogram showed an inverted P between R and T. It was clear that the tachycardia rapidly exhausted an already weakened myocardium.

In several of our patients, with a degenerate heart, attacks of paroxysmal tachycardia were associated with serious symptoms if the attack lasted for long, or as age increased.

Case M. A man who was recognized to have paroxysmal nodal tachycardia at the age of 60, had probably had his first attack at the age of 27, following typhoid fever. The attacks ceased for some years, but recurred at the age of 50, and continued. He had a high blood pressure and degenerate arteries. In his early life the paroxysms occasioned little discomfort, but as he grew older caused great exhaustion. He aged quickly, the attacks recurred every month, and when 63 an attack occurred and persisted, the pulse running 180–200 per minute until death ensued after 72 hours.

(2) *Continued Nodal Tachycardia.*—In a small number of cases nodal tachycardia has been observed in patients suffering from an infective disease, most commonly acute rheumatism and " bacterial " endocarditis. The onset of the rhythm may be preceded by nodal extrasystoles.

Case N. A man, aged 27, was admitted into hospital in 1922, suffering from acute rheumatism. Both the mitral and the aortic valves were affected, but he made a good recovery and resumed his work. In 1925 symptoms of cardiac failure ensued, followed by arthritis,and his progress was unsatisfactory ; the congestive symptoms increased, many veins became thrombosed, and infarcts occurred in the lungs and spleen. He died in February 1926, evidently with a " bacterial " endocarditis. During his last illness his fever was never high, not exceeding 101° F. His pulse at first was regular, numbering about 100 per minute. But five days before his death it rose to 150–160, and continued so until the end. The electrocardiogram showed a nodal rhythm, an inverted P appearing between S and T (Fig. 17).

Case O. A man, aged 42, was admitted into hospital on the fifth day of an acute pneumonia. His pulse was frequent and regular save when interrupted by an extrasystole. Next day there was a regular nodal rhythm, with a hidden P at the rate of 144 (Fig. 18). Next day his auricles were fluttering, and continued to flutter until

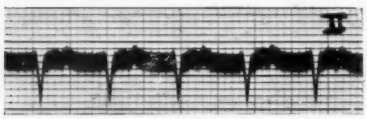

FIG. 17.—Nodal rhythm. Group 2. Case N. An inverted P is visible between S and T. Lead II.

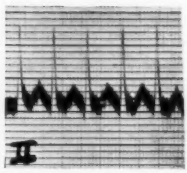

FIG. 18.—Nodal rhythm. Group 2. Case O. P is lost in the ventricular complexes. Lead II.

his death on the ninth day of illness. The mitral and aortic valves showed chronic endocarditis, with superadded acute re-infections. The myocardium showed an extensive patchy fibrosis. The nodes, unfortunately, were lost.

Cowan *et alii* (1913) reported six cases of acute endocarditis whose polygraph tracings showed a short *a.c.* interval. Microscopic examination disclosed involvement of the *a.v.* nodes in acute inflammatory lesions. The cardiac rhythm in these patients was regular, save for occasional extrasystoles, the maximum rate in the several cases being 105, 120, 140, 140, 140, 145 per minute.

Hume (1914) reported the occurrence of nodal rhythm during a small epidemic of diphtheria. A girl, aged 7, showed auricular extrasystoles on the ninth day of illness ; nodal rhythm with a pulse rate of 109 on the tenth and eleventh days ; auricular flutter on the twelfth day ; and a regular rhythm save for some auricular extrasystoles on the thirteenth day. She died on the fifteenth day of illness. A gross interstitial myocarditis was found, the *a.v.* node and bundle being unaffected. A boy, aged 7, had a normal rhythm on the seventh and eighth days of illness ; extrasystoles on the ninth day ; nodal rhythm with a pulse rate of 94 on the tenth day ; and 2–1 heart-block on the twelfth and thirteenth days. He died next day. The *s.a.* node was acutely inflamed and the *a.v.* bundle was engorged. The *a.v.* node, unfortunately, was lost.

(3) *Nodal Rhythm without Tachycardia.*—In this group the pulse rate is little altered, or less frequent than normal.

Case P. A gamekeeper, aged 55, was seen in 1914 for a strain of his right shoulder which had kept him off work for some weeks. He was a sparely built fellow, who looked younger than his years. He would not admit of any disability, and said that he could carry 14 couples of rabbits for a mile and a half without discomfort. His brachial arteries were thick and tortuous, but his heart seemed sound. His pulse numbered 75. The electrocardiogram showed normal ventricular complexes, and an inverted P, less than 0·10 sec. in front of R (Fig. 19). A fortnight later the rhythm was unaltered. He continued at work, but refrained from carrying heavy weights. A year later he was in good health. On this occasion the cardiac pacemaker was mobile, wandering between the sino-auricular and the auriculo-ventricular nodes,

though the pulse rate (69) did not alter. A normal rhythm, with P–R 0·14 in., changed within a couple of beats to a nodal rhythm with an inverted P, less than 0·10 in. before R (Fig. 20). The normal rhythm returned shortly afterwards, but the

FIG. 19.—Nodal rhythm. Group 3. Case P. P is inverted and precedes RS by 0·08–0·10 sec. Lead III.

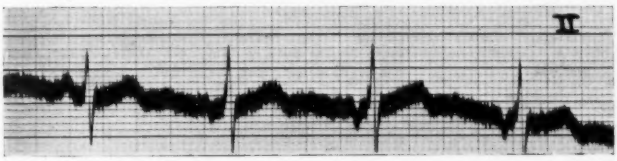

FIG. 20.—Nodal rhythm. Group 3. Case P. This record shows the transition from the sino-auricular to the auriculo-ventricular rhythm. Lead II.

change was not observed. In 1919, 1920, 1921 he was in good health, with a normal rhythm. In 1936, at the age of 76, he is still active as a grieve, and looks well and young for his age. His organs seem healthy and the cardiac rhythm is of sinus origin. An occasional extrasystole was missed in the electrocardiogram.

Boukspan (1928) reported the case of a locksmith, aged 29, who showed a nodal rhythm, with a pulse rate of 36–40, over a period of seven months. He had suffered from syphilis five years before, but the Wassermann test was negative. The first sound at the apex was " rough." On exertion his pulse rate rose to 44–46, and after atropine to 75. During the period of infrequent rate P was hidden, but when under the influence of atropine it reappeared, as a positive deflexion, before or after R. He continued at his work.

Wedd and Wilson (1930) reported the case of a man, aged 22, who had an apparently constant nodal rhythm. He had had a hemiplegic stroke at the age of 16, and was weak-minded. The mitral valve was stenosed. The usual pulse rate was about 60, but at times it fell as low as 36, and it sometimes failed for several seconds. This might occur spontaneously, or be induced by scolding or a sudden order. The pace-maker was not affected by atropine, or pressure upon the carotid sinus or the eyeballs, but the rate, when infrequent, was accelerated by exercise and atropine ; so the standstills were evidently vagal in origin.

Ledoux (1935) reported the case of a young farmer, aged 26, whose electrocardiograms invariably showed nodal rhythm, P appearing between R and T, over a period of eighteen months. His pulse rate was habitually infrequent,

30–60 per minute, and very variable. Syncopal attacks had recurred frequently when his pulse rate was low, but none was observed during his residence in hospital.

The following cases of nodal rhythm were under observation, at the time when the disordered rhythm was observed, on account of some form of cardiac disability.

Ritchie (1914) reported the case of a man, aged 72, with congestive cardiac failure, and high blood pressure. His pulse at first was grossly irregular with numerous nodal extrasystoles. On several occasions nodal rhythm supervened for an indefinite period, the pulse running in the nineties. Under treatment he improved, and the rhythm became normal. Another man, aged 47, was suffering from subacute myocarditis, without valvular lesions. For about a month a normal rhythm, with a pulse rate of about 90, alternated with a nodal rhythm, with a pulse rate of about 80. He improved for a time and resumed his work, but the improvement was transitory. His auricles went into fibrillation, and cardiac failure ensued. He died three and a half months after coming under observation. The cardiac muscle showed widespread fibrosis and cellular infiltration. The *a.v.* node and bundle were grossly involved.

White (1915) reported the case of a man, aged 37, who complained of obesity and weakness of the legs, probably due to early tabes dorsalis. His heart was normal in size and the sounds were closed and of fair value. His pulse on admission was 120, and there was a uricular flutter with defective conduction in the bundle branch (Type I). Ten days later the auricles were fibrillating. Four days later the rhythm was nodal, and this persisted for the next four months, the pulse rate falling to 40–43.

Matthewson (1915) reported the case of a man, aged 22, whose pacemaker frequently changed from the sino-auricular to the auriculo-ventricular node. He complained of shortness of breath and palpitation. His mitral valve was incompetent. His pulse numbered about 70 and the change of pacemaker recurred without any obvious change in the rate of the pulse, either node becoming dominant in an inconsistent fashion. When the rhythm was nodal compression of the carotid sinus failed to influence the pacemaker, but atropine and gentle exertion caused a return to normal rhythm.

A woman, aged 62, reported by Fussell and Wolferth (1920), had complained of attacks of palpitation since childhood, at first of little moment, but gradually increasing in frequency and in severity, till she had been forced to lead an invalid life for a couple of years. She had become short of breath upon exertion, and had noticed some œdema of the feet. After leaving hospital she died from cardiac failure. Her heart was slightly enlarged, and the mitral valve was incompetent. The attacks of palpitation occurred at any time, on exertion or during sleep. They could always be checked by holding her breath. They were due to an auricular tachycardia, and the pulse rate rose to 135 per minute. Her pulse rate was very variable, as the pacemaker shifted irregularly between the sino-auricular and the auriculo-ventricular nodes, the nodal rhythm persisting for a few seconds or as long as several minutes. The nodal rhythm might develop gradually, with decreasing P–R intervals, the rate gradually

slowing to 30–45 per minute ; or suddenly without any appreciable change in rate.

Richardson (1922) reported the case of a woman, aged 47, who was suffering from mitral disease and chronic nephritis. A normal rhythm, with a blood pressure of 150/98 mm., changed under the influence of digitalis to a nodal rhythm, with a pulse rate of 38–43 and a blood pressure of 180/120 mm. Atropine and pressure over the carotid sinus had no effect upon the pacemaker. She died eventually from pneumonia. The mitral and tricuspid valves were thickened, and the aortic valve acutely inflamed. Scars were present in the right auricle, and the ventricles showed a general fibrosis. The *s.a.* and the *a.v.* nodes were normal.

Conclusions

Causation of the Nodal Rhythms

1. *Paroxysmal Nodal Tachycardia.*—The nature of the paroxysmal change in rate is not clear. The sudden onset and termination of the paroxysms suggest a nervous cause, cessation of the controlling activity of the vagus, or overaction of the accelerator mechanism. But the failure of vagal stimulation to control the tachycardia, save in very exceptional cases, shows that the nervous control is temporarily lost, from some obscure cause.

2. *Continued Nodal Tachycardia.*—Continued nodal tachycardia has hitherto been observed only in cases of acute myocarditis, and the change in rhythm is presumably due to involvement of the auriculo-ventricular node in an inflammatory mass.

3. *Nodal Rhythm without Tachycardia.*—This group in which the pulse rate is unaltered or slowed evidently owns several causes. In some patients the heart is apparently sound, in others grossly diseased. In some the change of rhythm ensues during the exhibition of digitalis and ceases on withdrawal of the drug. In some the normal rhythm follows the administration of atropine, but in others the abnormal rhythm is unaffected. It thus seems clear that some cases are due to vagal overaction, whether spontaneous or induced by digitalis, and the consequent domination of the rhythm by the auriculo-ventricular node. But the cases which are not influenced by atropine must own a different cause, perhaps of myocardial origin.

Diagnosis of Nodal Rhythm

This can only be established by polygraphic or electrocardiographic records. Its occurrence can sometimes be suspected by the appearance of large pulsatile waves in the jugular veins, caused by the coincident contraction of the auricles and ventricles, closing the tricuspid valve and so entailing the reflux of the auricular blood into the veins when the right auricle contracts.

Prognosis of the Nodal Rhythms

1. *Paroxysmal nodal tachycardia* is not incompatible with long life. In patients whose hearts are sound, and cardiac reserve between paroxysms ample, the immediate and the ultimate outlook is good. If an attack persists for many hours symptoms of cardiac insufficiency may arise, but they rapidly subside on cessation of the tachycardia.

The outlook is, however, serious in patients whose hearts are unsound, as the tachycardia rapidly exhausts an already weakened heart ; and is more serious if symptoms of cardiac weakness already exist.

2. *Continued nodal tachycardia* always makes the outlook serious, as it seems to be indicative of acute myocarditis. Its occurrence in the cases hitherto reported has always been followed shortly by death.

3. *Nodal rhythm without tachycardia* with an unaltered or a slowed pulse rate has a variable outlook according to the cause. It is serious in the cases where the alteration in rhythm is due to a myocardial lesion and in those due to digitalis, as the exhibition of the drug predicates some pre-existing cardiac difficulty. In the nervous cases the outlook is good.

V. SUMMARY

Alterations in the rhythm of the heart, as isolated signs, are not necessarily of serious significance.

James Mackenzie stated (1912) that *sinus irregularity*, in a healthy individual, was a normal occurrence, and many observers have subsequently confirmed the accuracy of his dictum. It is true that *sino-auricular block* may, as shown in some of the cases which we have collected, be accompanied by serious symptoms, but the subsequent history of these patients shows that, in the absence of signs of cardiac disease, the prognosis from the cardiac standpoint is quite good. Any danger lies in the nature of the nervous disorder which occasions it.

The occurrence of *extrasystoles, per se*, has not any sinister significance. It is true that if they recur rapidly for long periods the mere rapidity of the cardiac contractions may produce cardiac failure, but, in the absence of signs of cardiac disease, any cardiac symptoms rapidly pass as soon as the normal rhythm is restored. A man, who died from paralysis agitans at the age of 75, had been liable to attacks of paroxysmal tachycardia from the age of 18, and had lived a very strenuous life for many years.

The prognosis in cases of *nodal rhythm* depends, as in sinus irregularity, upon the nature of its cause. The outlook is serious if it is due to myocardial lesions ; but good if it is due to nervous causes.

The occurrence of *auricular fibrillation* is generally a signal of impending danger, but the exceptions to the rule are fairly numerous. Cases have been reported where the arrhythmia persisted for ten or even twenty years. One of my patients bore a child safely although her auricles had been in fibrillation for at least three years. The irregularity is not the important factor in the

failure. The danger lies in the frequency of the ventricular contractions or in the continued activity of the cause of the irregularity. Fibrillation may be due to several causes : pathological lesions in the auricular muscle, of chronic or acute character ; auto-intoxications, as in Graves' disease ; poisons introduced from without (digitalis, anæsthetics, coal-gas poisoning, etc.) ; physical stresses ; and perhaps to disturbances of the nervous control of the heart (Cowan 1929). The cause may be permanently or temporarily in action. The prognosis follows the cause.

Heart-block and bundle branch block repeat the story. As they are most frequently due to myocardial disease the prognosis is, as a rule, serious, but many patients live in fair health for many years. Again the danger lies in the rate of the ventricular contractions, or in the character of the process which has disturbed the rhythm of the heart.

This paper is based upon electrocardiographic records, upon what may be called laboratory methods of diagnosis. But it seems to me that there is a tendency at the present time to overestimate the value to clinical medicine of such methods. The wood tends to be hidden by the trees, and an attempt is made to treat a symptom, or even a sign, and not a sick man.

An irregular pulse is a useful signpost, but the causes of irregularity are numerous, and not necessarily disease of the heart. In many cases treatment, if it is to be helpful, must be based upon the general picture of the illness, and directed to regions outwith the heart.

I have great pleasure in acknowledging the kindly assistance of many friends ; in particular G. B. Fleming, A. G. Gibson, I. G. W. Hill, and E. E. Laslett.

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DIGITALIS IN HEART FAILURE WITH NORMAL RHYTHM

BY

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In the first edition of his *Diseases of the Heart*, published in 1908, Mackenzie said : " the nodal rhythm is present in the majority of cases of severe heart failure and in a great many the immediate breakdown is directly attributable to the inception by the heart of this abnormal rhythm." In the third edition, in 1913, the term " auricular fibrillation " was substituted for that of " nodat rhythm," the researches of Lewis and others having established their identity. Mackenzie gave the proportion as " 60 or 70 per cent. of all cases of serious heart failure met with in practice." It is even now generally admitted that auricular fibrillation affects the majority. Lewis (1937) has said that at least 70 per cent. of all cases of failure with congestion display fibrillation.

To quote Mackenzie (1911) on digitalis : " Cases of auricular fibrillation are more readily and more markedly affected than cases with normal rhythm." Dissentient views began to be expressed perhaps first by Janeway (1913), and later especially by Christian (1919) whose paper was entitled " Digitalis therapy : satisfactory effects in cardiac cases with regular rhythm." In a further study (Christian, 1922) he affirmed that : " Digitalis is fully as effective in the treatment of chronic cardiac cases without auricular fibrillation as it is in those with fibrillation." Luten (1924) reached the same conclusion. Yet most authors to-day admit that the beneficial effect is greater in auricular fibrillation, though they do not deny (and Mackenzie did not) that it is also effective in heart failure without fibrillation. Lewis's experience is that occasional cases presenting regular heart action respond favourably (Lewis, 1937).

We have spoken as if auricular fibrillation occurred indiscriminately in heart failure and as if the response to digitalis depended simply on the presence or absence of this arrhythmia. But it might be that fibrillation connoted a different group of cardiac lesions and thereby a different reaction to digitalis. It was with this idea in mind that Parkinson and Clark-Kennedy (1926) undertook an inquiry into heart failure with normal rhythm. It was established that there is an important relation between the pathological basis for a cardiac disease and failure and the presence or absence of fibrillation. In general,

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† Received Nov. 19, 1938.

auricular fibrillation was the rule in rheumatic heart disease and goitre, while failure with normal rhythm was the rule in hypertension, coronary disease, and emphysema.

Apart from the factor of rhythm and the factor of a specific pathology conditioning rhythm, there is a third factor of the *heart-rate* during failure. A regular rhythm failure may occur with a normal or moderate ventricular rate, or a high rate. This has been much discussed (Mackenzie, 1911 ; Sutherland, 1919 ; Christian, 1922 ; Hay, Jones, and Ince, 1927 ; Marvin, 1927 ; and Luten, 1936 *a*, *b*). Apart from its pharmacological interest, the rate in failure with normal rhythm might indicate where digitalis acts beneficially, e.g. those with a high rate might respond and those with a normal rate might not respond.

The question we have placed foremost in this inquiry is how often and how far digitalis can help in a clinical sense patients who are suffering from heart failure without auricular fibrillation. It was hoped also to learn something from the kind of failure, the ætiology, the pathological basis, or the ventricular rate, which would aid in selecting the patient most suited for digitalis treatment. This clinical discrimination in the use of digitalis in heart failure with normal rhythm should soon be settled, because a mercurial diuretic will in future so often be employed alone or along with it that the separate effect of each should be established.

Method of Investigation

Our observations were made chiefly on patients admitted to the beds of the Cardiac Department of the London Hospital over a period of eighteen months ending July 1938. Every patient considered to be suffering from congestive heart failure with normal rhythm was admitted for therapeutic observation. A combination of two or more of the following criteria was required : obvious dyspnœa, œdema, enlargement of the liver, congested lungs with or without hydrothorax, and venous distension in the neck. Left ventricular failure with paroxysmal dyspnœa and without systemic congestion was excluded from this inquiry. The degree of failure was assessed as slight or as moderate to severe. An electrocardiogram was taken. The chest was examined by X-rays and by teleradiograph when necessary. If a hydrothorax was present, teleradiograms were taken at intervals to register progress. The Wassermann reaction was tested except in a few of the rheumatic group. The blood urea was estimated and primary renal disease was excluded clinically and occasionally by necropsy.

For a period of at least a week no active treatment was ordered and the patient simply remained in bed. If digitalis had been given before admission, this control period was extended. A few patients were so ill on admission that strophanthin or salyrgan had to be given intravenously ; if so, an additional period without drugs was arranged. But if at about the fifth day of rest alone a patient was worse, it was felt unnecessary to withhold digitalis any longer for control purposes. A sedative (usually morphine) was given at night if required. Fluids were not often restricted and so the disturbing factor of another therapeutic measure was avoided. It proved that 40 oz. (1,137 c.c.) daily was

seldom exceeded, but in a few where fluid was restricted to 30 oz. (852 c.c.) the restriction was equally applied during the rest and the digitalis period Frequent clinical notes were made in order to assess the rate and the extent of any improvement in the symptoms and signs of failure. During the control period we had to reject a number of cases because of pyrexia or other complications such as pulmonary infarction. Sometimes rest in bed alone was so beneficial that the residual failure was incompatible with adequate observations on digitalis action.

The Digitalis Course

Digitalis was administered in friable tablets containing one grain of the powdered leaf, pulvis digitalis folia, B.P., which is standardized biologically to contain 10 international units per gramme. This preparation is identical with that in the United States Pharmacopœia of 1936. The usual dose was 2 grains three times a day (6 grains or 0·4 g. daily). In the early cases 1 grain three times a day was given for 14 days, but its effect was less easily demonstrated. Sometimes 3 grains three times a day were given and then usually in the more resistant cases or at a subsequent test. The normal period of digitalis administration was 7 days. It was found that 2 grains three times a day was a practicable dose and rarely led even to slight toxic effects, and then only towards the end of the test.

Certain criteria were considered for determining the result of rest in bed alone and of the digitalis course. Little attention was given to the patient's feelings expressed in response to the usual polite enquiries, for these are so much influenced by the patient's good will. Observed dyspnœa at rest or on slight movement was important, as were also the degree of distress and obvious discomfort including cough, insomnia, epigastric (hepatic) discomfort, and flatulence bound up with failure. The rate of the respiration was charted, but it seemed little related to the degree of dyspnœa as felt by the patient or observed by the physician. As Christian (1922) said : " Dyspnœa is poorly, sometimes not at all, depicted by the charted rate of respiration." Crepitations at the bases of the lungs were not of much value, as they often disappeared with the first few days of rest. Hydrothorax always figured in the rating, but its place and importance would scarcely have been realized had X-ray examination of the chest in failure not been practised. So objective a sign as œdema must always be an important index of progress. The size of the liver was almost as important. Diuresis and loss of weight were valuable criteria. The venous distension did not prove generally serviceable as a guide to the progress of improvement, and the same may be said of cyanosis.

Digitalis Effect or Digitalis Benefit.—The result of the digitalis course might be considered either as a pharmacological effect or as therapeutic benefit. Fall in heart-rate was not here regarded as an indication of therapeutic benefit, and this policy enabled us to observe the relation of slowing to benefit as assessed by our criteria of improvement, e.g. loss of œdema.

In most patients only one course of digitalis was studied ; but in a few cases of persistent failure two courses of digitalis were observed during the stay in

hospital. When further observations were made during a subsequent admission, the patient was regarded as a new case.

Failure with Auricular Fibrillation.—Although this paper mainly concerns failure with normal rhythm, we have also studied in like manner thirty cases of failure with auricular fibrillation for comparison. The ventricular rate was counted at the apex daily throughout both the control period and the digitalis period.

Salyrgan.—In several cases salyrgan, 2 c.c., was given intravenously either immediately following the end of the digitalis course or else a few days later, so as to judge whether a diuresis was possible by means other than digitalis. Neither ammonium chloride nor ammonium nitrate was used as an adjuvant.

Classification of Cases

The choice of cases was made primarily on the presence of cardiac failure with normal rhythm, and without regard to the ætiology of the causal hear disease. Hypertensive, rheumatic, and syphilitic heart disease together formed the bulk of the cases (Group 1–4 ; Table I). Of the ten syphilitic cases, all had aortic incompetence. Group 5 included atheromatous aortic incompetence and coronary disease without hypertension or cardiac infarction. No typical example of heart failure from emphysema was found.

There were 22 observations on 18 patients with heart failure and normal rhythm who are classified among subsidiary groups, namely: congenital heart disease, acute rheumatic carditis with valvular disease, infective endocarditis, complete heart-block, and chronic nephritis. They were treated just the same, but they were patients in whom digitalis would not usually be employed or in whom its action would be less promising. Thyrotoxic heart disease was excluded.

Previous Observations

The main question is how far digitalis can benefit patients with failure in normal rhythm ; and the further question is the comparable benefit in failure with auricular fibrillation. Though there are a large number of statements based only on personal experience and conviction, there are a limited number of detailed studies of these questions. The largest series are those of Christian (1922), Luten (1924), and Marvin (1927).

Christian contrasted 40 cases of normal rhythm with 57 cases of auricular fibrillation. Though without preliminary control periods, there is good evidence of successful treatment which was independent of rhythm. In both classes, his high percentage of successes is augmented by the inclusion of a reduced heart-rate as an indication of success. Possibly, too, the restricted fluid favoured his results.

Luten studied 20 cases of failure with normal rhythm. After control observation, he gave digitalis in massive doses (Eggleston method) and found that " digitalis affected most of the cases favourably." These results applied to failure from chronic myocarditis with or without hypertension or œdema.

No cases with auricular fibrillation were used for comparison, but the conclusion was that " patients with myocardial insufficiency improve under proper digitalis administration in about the same proportion of cases as do patients with auricular fibrillation."

Marvin investigated 20 cases of advanced congestive failure with much œdema, paying particular attention to ætiology in relation to the digitalis response. Eggleston's method of administration was used after a control period of observation. Benefit from digitalis followed in 11 of the 20 cases ; none in the rheumatic group of 5 ; 3 in the syphilitic group of 5 ; and 8 in the arteriosclerotic group of 10 ; but only two cases (both in the last group) showed great improvement comparable with that seen in auricular fibrillation. Slowing occurred once only in each group.

Mackenzie (1911) was not impressed with digitalis effect in failure with normal rhythm, though he recognized that digitalis might on occasion produce a satisfactory diuretic response and independently of the heart-rate. He laid great stress on the beneficial effect of rest in bed when the rhythm was normal, and attributed to it much of the apparent digitalis response.

Hay, Jones, and Ince (1927) gave digitalis tincture until toxic symptoms appeared in nine controlled cases of failure with normal rhythm. Four cases showed benefit, the greatest being seen in rheumatic valvular disease with œdema. They found that fall in heart-rate was not necessarily accompanied by subjective improvement. Kellum (1932) studied digitalis diuresis in twelve controlled patients with failure and œdema, with one exception normal rhythm being present. Diuresis was moderate in three and slight in four cases, but fluids were restricted to an average of 770 c.c. (27 oz.) per day. The pulse was reduced in one-half the cases to an extent of an average of thirteen beats per minute. In an analysis of case records of failure with sinus mechanism, Hyman and Fenichel (1932) found that moderate doses of digitalis improved only 4 of 13 arteriosclerotic patients and none of 9 in the rheumatic group. Reduction in heart-rate itself was reckoned as evidence of improvement. In the presence of auricular fibrillation, digitalis helped 22 of 27 arteriosclerotic and all 30 rheumatic cases. Nineteen ambulatory patients with paroxysmal dyspnœa and normal rhythm were studied by Harrison, Calhoun, and Turley (1931). Sixteen cases were completely relieved by digitalis. As paroxysmal dyspnœa is a special product of left ventricular failure, we regard this result as of great practical importance. While investigating the effect of digitalis in over 100 cases of pulsus alternans in sinus rhythm, Windle (1917) opined that it could be given " with the confidence in a first attack of dropsy that it will almost surely do good." In an earlier paper dealing with rheumatic heart disease, Windle (1913) concluded that " digitalis often acts as well, as certainly, and as speedily on the general symptoms of heart disease with dropsy and regular pulse as it does in like cases of auricular fibrillation." In children under 14 with rheumatic infection and failure with normal rhythm, Sutherland (1919) concluded that the digitalis in rapid heart action was as specific in reducing the heart rate as in auricular fibrillation and that the beneficial effects were as striking. When the pulse-rate rose it could be repeatedly lowered by digitalis.

The general conclusion of these investigators is that digitalis is indicated in heart failure with normal rhythm and that it often succeeds, though not so often and seldom so well as in heart failure with auricular fibrillation. Christian (1922) and Luten (1924) are exceptional in claiming that it succeeds as frequently and as well in normal rhythm as in auricular fibrillation. Opinions expressed by West and Pratt (1920), Robinson (1923), Fraser (1924), Robinson, White, Eggleston, and Hatcher (1924), Cushny (1925), Levy and Mackie (1927), Bramwell (1937), and East (1937) support the general conclusion, which is also the teaching found in current books on cardiology, e.g. Lewis (1937), White (1937), Fishberg (1937), East and Bain (1936), Levine (1936), Cowan and Ritchie (1935), and Harrison (1935).

In French reports the rhythm is not much considered in this relation, and Laubry (1938) says that digitalis is suitable for all cases of cardiac failure. Among German books, Scherf (1938) says that quite good digitalis action can be observed in hearts in failure without fibrillation because of the drug's action on the contractile power of the heart muscle, and that slowing of the heart-rate need not be produced. The most striking results occur in fibrillation, when the rate is rapid because slowing is produced. If the heart-rate is between 70 and 80, the conditions are closer to those with normal rhythm.

Results Obtained in this Series

There were 47 patients, 36 males and 11 females, on whom 58 tests on digitalis effect were carried out. In the great majority (42 of 47) the failure was moderate or great on admission. The patients were divided into five groups and the group distribution is indicated in Table I.

For purposes of comparison there were 30 cases of failure with auricular fibrillation on whom 32 tests were made. Again the majority (22 in 30) were in moderate or great failure on admission. As expected, the association being rare, there were no syphilitic cases with fibrillation. Unlike the normal rhythm series where each ætiological group (except Group 5) is well represented, almost half the fibrillating cases were rheumatic.

Table I

RESULTS OF DIGITALIS TREATMENT IN HEART FAILURE (1) WITH NORMAL RHYTHM, AND (2) WITH AURICULAR FIBRILLATION

Normal Rhythm

GROUP	BASIS OF FAILURE	NUMBER OF PATIENTS	NUMBER OF TESTS	CLINICAL IMPROVEMENT (IN TESTS)	REDUCTION IN RATE (IN TESTS)
1.	Hypertension				
2.	Hypertension and past Coronary Thrombosis	25	32	19	14
3.	Chronic Rheumatic Valvular Disease.	11	14	10	7
4.	Syphilitic Heart Disease.	9	10	5	6
5.	Unclassified.	2	2	1	0
	Total	47	58	35	27
	Percentage of Tests ..			60·3%	46·5%

TABLE I—*continued*

AURICULAR FIBRILLATION

GROUP	BASIS OF FAILURE	NO. OF PATIENTS	NO. OF TESTS	CLINICAL IMPROVEMENT (IN TESTS)	REDUCTION IN RATE (IN TESTS)
1.	Hypertension	12	14	9	10
2.	Hypertension and past Coronary Thrombosis				
3.	Chronic Rheumatic Valvular Disease.	14	14	12	14
4.	Syphilitic Heart Disease	0	—	—	—
5.	Unclassified.	4	4	2	4
	Total	30	32	23	28
	Percentage of Tests ..			71·9%	87·5%

The good effect of rest in bed without other treatment is discussed later, the influence of this on the pulse-rate, weight, and balance of fluid intake and urinary output in one patient is shown here in Fig. 1.

Of the 58 tests on digitalis effect in normal rhythm, 35 (60%) were successful, there being 17 (29%) with slight benefit and 18 (31%) with moderate or great benefit. Only 7 (12%) showed striking improvement and a composite chart is given of four of them (Fig. 2). In this chart the dose is given as gr. ii, t.i.d. correctly for three of the cases, but the fourth had gr. iii, t.i.d. ; a similar

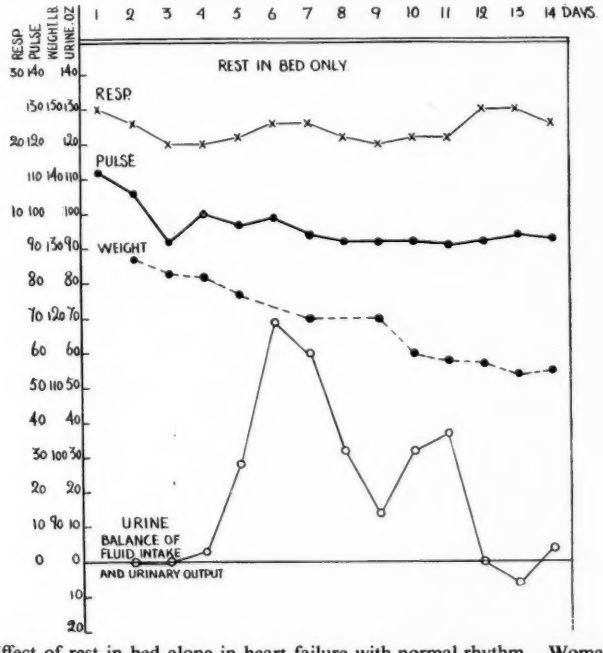
1 2 3 4 5 6 7 8 9 10 11 12 13 14 DAYS
RESP. PULSE WEIGHT LB. URINE OZ.
REST IN BED ONLY
RESP.
PULSE
WEIGHT
URINE
BALANCE OF FLUID INTAKE AND URINARY OUTPUT

FIG. 1.—Effect of rest in bed alone in heart failure with normal rhythm. Woman, aged 52, with hypertensive heart disease, gross œdema, and hydrothorax.

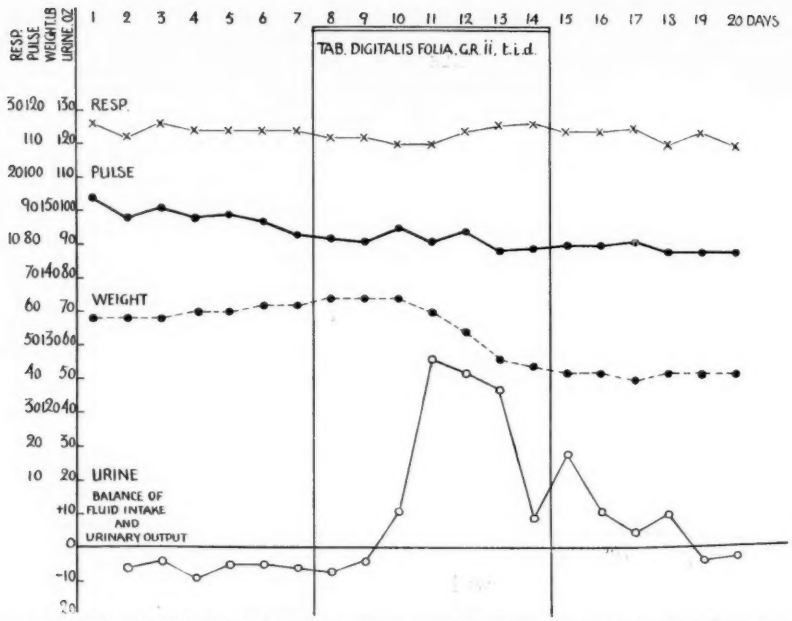
RESP.
PULSE
WEIGHT LB
URINE OZ
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 DAYS
TAB. DIGITALIS FOLIA, GR ii, t.i.d.
RESP.
PULSE
WEIGHT
URINE
BALANCE OF FLUID INTAKE AND URINARY OUTPUT

FIG. 2.—Effect of digitalis in heart failure with normal rhythm. A composite chart from four severe cases, all with œdema.

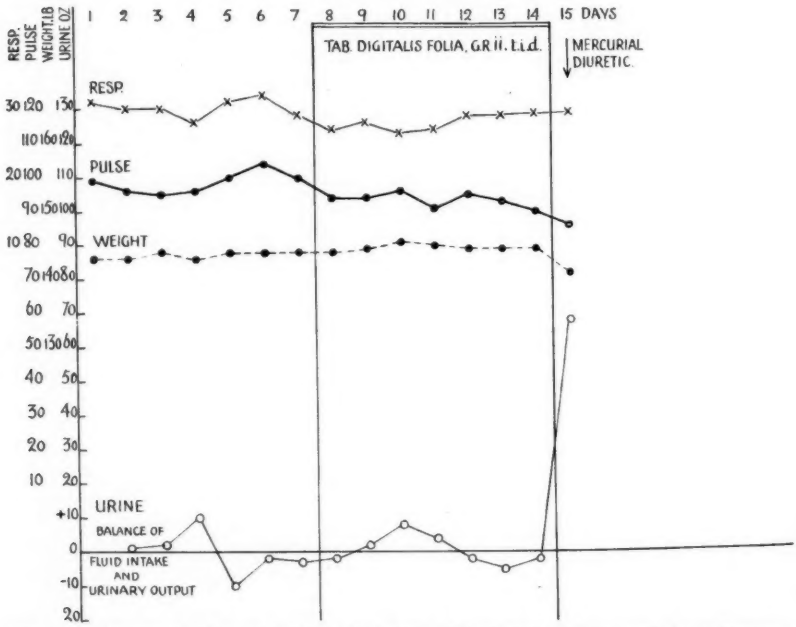

FIG. 3.—Absence of effect of digitalis in failure with normal rhythm, and the subsequent effect of a mercurial diuretic. A composite chart from four severe cases, all with œdema.

exception holds for Fig. 3, which shows the complete ineffectiveness of digitalis in four cases. These results are rather disappointing, especially with regard to the infrequency of striking benefit, for only about one in eight patients in failure with normal rhythm can be expected to respond thus well. Nevertheless the majority benefit to some extent and would probably benefit more from administration over long periods.

In the series with auricular fibrillation, some degree of benefit from digitalis was seen more often, though not much oftener than in normal rhythm (72% against 60%). The incidence of great improvement was however more than twice that in normal rhythm (28% against 12%).

Diuresis occurred in about half of all test courses in normal rhythm (27 in 58) ; it was moderate in eight and striking in six (10·3%). The majority (24 of 35) of the observations with benefit were, as expected, accompanied by diuresis.

In auricular fibrillation diuresis occurred in 14 of 32 tests, much as in normal rhythm. Diuresis was moderate in eight and striking in four (12·5%). Of 23 with benefit, 16 showed diuresis.

Successive Courses of Digitalis in the Same Individual.—When a patient in failure with normal rhythm benefits from digitalis on a first occasion, he is likely to respond again when in failure some weeks or months later : six of eight patients responded thus. If digitalis fails at the first test, it does not follow that it will fail again ; indeed, of four failing to respond at the first, two responded at a second test when again in failure.

Factors which may Influence Digitalis Response

(*a*) *Rest in Bed.*

The effect of rest in bed during the control period was evaluated in the 47 cases with normal rhythm. Benefit occurred in 17 (about 40%) ; in 9 it was moderate or great, and in 1 of these it was outstanding (Fig. 1). Nine patients, six of them with hypertension, became worse during the rest period. Of these nine, five were considerably improved by digitalis, in three the immediate downward trend of failure was arrested, while the remaining patient became worse on digitalis and died.

Mackenzie (1911) and Janeway (1913) having drawn attention to the effect of rest, most subsequent investigations on digitalis have been controlled in this way. But rest also remains a factor in enhancing digitalis effect during the course. If progress has not become stationary on rest alone, then the " rate of progress " (Luten, 1924) becomes important when assessing the benefit from digitalis.

Contrary to what was expected, benefit from rest in bed was only a little less frequent in the fibrillating cases. All the seven patients (four in the rheumatic group) who deteriorated on rest in bed benefited from digitalis.

(*b*) *The Nature of the Heart Disease.*

No great difference was found in digitalis response between the various ætiological groups with normal rhythm, though the rheumatic group responded best. In fibrillation, however, the rheumatic group responded far better than any other (Table I). Excepting the rheumatic group, the separate ætiological groups with fibrillation showed little better results than the corresponding groups with normal rhythm.

(*c*) *The Heart-rate.*

The heart-rate is not usually very frequent in failure with normal rhythm. In 47 patients on admission, the average was 91, and at the end of the control rest period it had fallen to 86. In the rheumatic group (which showed most improvement) the average rate was only 82. Table II shows that digitalis reduced the heart-rate in about half of the 58 tests (46%). Benefit resulted in two-thirds (18 of 27) of those with and in about half (17 of 31) of those without slowing. Considerable slowing (over 15) was rather more often accompanied by clinical improvement than was slight slowing (below 15) ; and in each of five cases with a great reduction in rate (over 30), benefit always occurred. Slowing, especially if great, is therefore a promising sign though it is not necessarily followed by clinical improvement ; and improvement is by no means dependent on slowing, as Figs. 2 and 3 indicate.

TABLE II

THE EFFECT OF DIGITALIS ON THE HEART-RATE IN HEART FAILURE (1) WITH NORMAL RHYTHM, (2) WITH AURICULAR FIBRILLATION

	NORMAL RHYTHM	AURICULAR FIBRILLATION	
		NON-RHEUMATIC	RHEUMATIC
No. of cases	47	16	14
No. of observations..	58	18	14
(*a*) *With reduction in rate*	**27**	**14**	**14**
	(46%)	(78%)	(100%)
Rate before digitalis	85	93	108
Rate after digitalis	67	70	70
Average fall in rate	18	23	38
Benefit from digitalis	18	9	12
	(67%)	(64%)	(86%)
No benefit from digitalis	9	5	2
	(33%)	(36%)	(14%)
(*b*) *Without reduction in rate*	**31**	**4**	**0**
	(54%)	(22%)	
Rate before digitalis	85	89	—
Benefit from digitalis	17	0	—
	(55%)		

Some authors (Lewis, 1919 ; Sutherland, 1919 ; Eggleston, 1920 ; Cushny, 1925 ; Levy and Mackie, 1927 ; and Fishberg, 1937) have held that digitalis

is more likely to induce slowing if there is tachycardia. We agree in part, for four of the five cases above mentioned with a striking reduction in rate had a high initial rate (over 90). But, in general, slowing was as common when the initial rate was near the normal as when it was higher.

No relation between the ætiology of failure in normal rhythm and slowing from digitalis was discovered ; and slowing in the rheumatic group was not more often seen than in the other groups (Table I). Extensive myocardial damage need not prevent a fall in rate, for the group of hypertension with past coronary thrombosis showed the same incidence of slowing as the other groups. In three of seven cases with bundle branch block, digitalis reduced the rate.

Unlike Cohn (1915) and Eggleston (1920), we found that slowing, whether slight or great, was more common in the absence than in the presence of œdema (70 per cent. against 40 per cent.). Yet slowing was common enough among the œdematous. Digitalis diuresis had no relation to the fall in rate, nor had the severity of failure.

The Rate Factor in Failure with Auricular Fibrillation.—The average rate on admission for all cases, again in Groups 1 to 5, was 111, 20 points higher than the corresponding figure in normal rhythm. Rest alone produced an average fall of 10. Digitalis caused a reduction in rate in 28 of 32 observations (87·5%), and of these 75 per cent. showed benefit. The average fall was 30. Of the remaining four cases which showed no slowing, none were improved. The rate factor was evidently more important in auricular fibrillation than in normal rhythm ; for the initial rate was higher, slowing by digitalis was much commoner and in its absence no benefit accrued.

Looking at the ætiological groups, rheumatic valvular disease stood out in many respects : the rate on admission was 121 (148 if no digitalis had been given before admission) ; rest induced a fall of 13 ; digitalis invariably reduced the rate with an average fall of 38 ; and 86 per cent. derived benefit. Considering the rate factor within the rheumatic fibrillation group, it was the patient with a rapid ventricular rate of over 100 who responded best to digitalis (Fig. 4). Even if we took those with a rate below 100, the response to digitalis was distinctly better than in non-rheumatic fibrillation, i.e. 72 per cent. against 50 per cent. But it was no better than in rheumatic cases with normal rhythm and a comparable rate of 81 and 82, i.e. 72 per cent. in each. Digitalis may act well in older patients (over the age of 60) in failure and fibrillation with an infrequent rate (under 80), a group to which some attention has been given by Mackenzie (1911) and Fraser (1928) ; we had five such cases, two of whom not only improved greatly, but showed the greatest reduction in rate—16 and 20.

Table II analyses the rate factor. The non-rheumatic cases of fibrillation occupy an intermediate position between those with normal rhythm and the rheumatic cases with fibrillation. Despite the greater slowing, non-rheumatic fibrillation was no more helped by digitalis than was normal rhythm. The rheumatic group with fibrillation alone justifies the prominence given to the rate factor in digitalis response in failure. This, too, is the group where digitalis can exert its well-known power of controlling the rate and so of prevent-

ing the recurrence of failure. No one claims that digitalis has the same value either in controlling rate or in warding off failure when the rhythm is normal ; though it should, of course, be used with these objects in view.

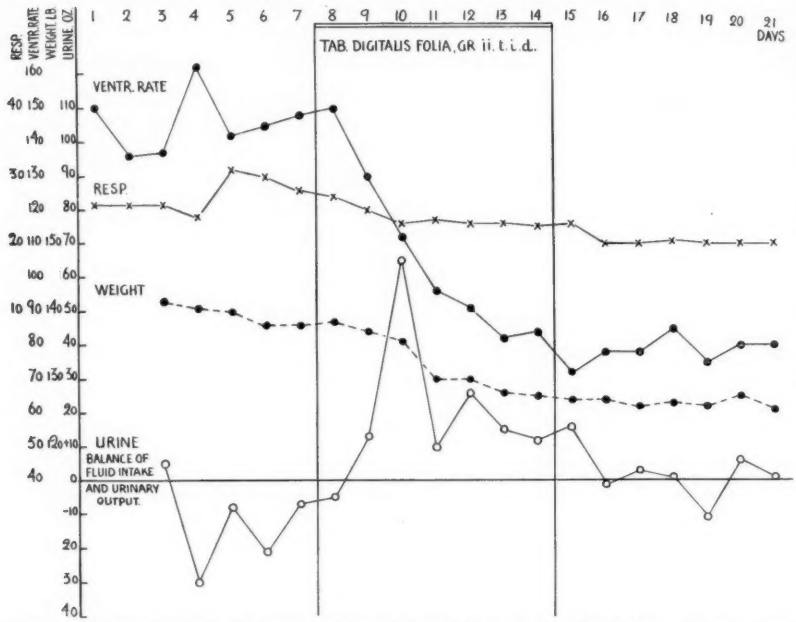
RESP.
VENTR. RATE
WEIGHT LB
URINE OZ
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 DAYS
TAB. DIGITALIS FOLIA, GR ii. t. i. d.
VENTR. RATE
RESP.
WEIGHT
URINE
BALANCE OF FLUID INTAKE AND URINARY OUTPUT.

FIG. 4.—Effect of digitalis in rheumatic heart failure with auricular fibrillation. Woman aged 64 with mitral stenosis, great œdema, and hydrothorax

(d) Œdema.

We included cases without demonstrable œdema at the end of a control period (most had œdema on admission), provided there remainded ample evidence of failure ; and digitalis was by no means without effect upon them. In fact, considerable benefit was obtained as often without as with œdema, and some response occurred in 75 per cent. without œdema against 52 per cent. with œdema. Figures for the fibrillation series ran parallel.

Though with normal rhythm considerable diuresis was twice as common in patients with œdema, yet some diuresis was not uncommon without œdema, 6 in 16. Thus we cannot go so far as some (Hay, Jones, and Ince, 1927) who have stated that no œdema means no diuresis. In patients with cardiac failure, several pints of fluid may be retained in the tissues before œdema becomes manifest, and a digitalis diuresis reveals what has really been latent.

In the auricular fibrillation series without œdema, considerable diuresis was even more common than in normal rhythm without œdema (6 in 12 against 2 in 16).

(*e*) *Hydrothorax. Respiratory Rate.*

There were 13 normal rhythm cases with hydrothorax ; in six it was on the right, in four on the left, and in three bilateral. The rheumatic group was not represented ; syphilitic heart disease accounted for three (one right, two left) ; and the remainder were in the two hypertension groups. Digitalis dispelled the hydrothorax in five and reduced it in one. Though the hydrothorax was not appreciably affected in three patients, they derived benefit from the drug in other directions, e.g. loss of œdema. Patients with hydrothorax responded like those without.

It was remarked that even with hydrothorax the charted rate of respiration immediately before digitalis was in some cases as low as 20, the average being 25, as it was also in severe failure without hydrothorax. With improvement on digitalis the respiratory rate usually fell, though the average fall was only two or three ; sometimes there was no fall (Fig. 2).

In five cases of auricular fibrillation with right hydrothorax, digitalis was effective, the hydrothorax being dispelled completely in two and reduced in one.

Thus in severe failure, whatever the rhythm, digitalis is as likely to benefit a patient with a hydrothorax as one without.

(*f*) *The Duration of Incapacity.*

It might have been thought that in cases with normal rhythm and failure of short standing (under 6 months), the immediate results from digitalis would be better than in those of longer standing, but this idea was not supported by our figures, any more than the idea that patients in a first failure would respond better than in a subsequent failure. The same observations apply to auricular fibrillation.

(*g*) *The Severity of Failure.*

This did not much matter in deciding whether digitalis would act or not. Fourteen of 20 tests in slight failure and 17 of 38 in moderate or great failure were successful. In fibrillation the proportions were 8 of 12 in slight failure, and 15 of 20 in moderate or great failure.

The superiority of digitalis in auricular fibrillation is most evident if the severer grades of failure are alone considered. Only 7 of 38 in normal rhythm responded brilliantly, but no fewer than 9 of 20 in auricular fibrillation.

(*h*) *Age and Prognosis.*

The age factor arises chiefly in relation to ætiology. The rheumatic cases with normal rhythm were on an average 22 years younger and the syphilitic group on an average 8 years younger than the hypertension groups, where the average age was 61. The age factor proved to be of little importance, though the tendency was for a decline in digitalis response as age advanced. Yet among those aged 65 to 70, no fewer than five of nine patients responded and two of them well.

The pathological basis of failure could not be correlated with the likelihood of digitalis response. In auricular fibrillation, however, the incidence of benefit was far greater in the rheumatic group than in the non-rheumatic groups, and this has been discussed above, under '*c*'.

We thought it likely that in patients with normal rhythm, whose survival-period proved to be under one year, digitalis might be less effective. This did not prove to be the fact, for of 18 patients who died within a year, 10 had benefited from digitalis and 8 had not. Moreover, of four who died within a few weeks of uncomplicated heart failure (proved at necropsy in three), two responded well to digitalis, a proportion not very different from those with a better prognosis. There was no essential difference in the death rate between the ætiological groups in normal rhythm, and we know that 18 of 29 patients who were followed up to July 1938 died within a year.

Comparison of Digitalis Effect during Normal Rhythm and during Auricular Fibrillation in the Same Individual.—There were five such cases, in two of which the fibrillation was produced or more likely precipitated by digitalis. It happened that the average rate was the same in normal rhythm as in auricular fibrillation, *i.e.* 88. Under digitalis during normal rhythm, the rate fell to 81, while during auricular fibrillation the rate fell to 66 : a fall of 7 and 22 respectively. As to clinical benefit, we have adequate observations on four of them, and in two digitalis failed both in normal rhythm and in auricular fibrillation. In the other two digitalis gave a better clinical result when fibrillation obtained.

Digitalis and Mercurial Diuretics

When a course of digitalis failed, a mercurial diuretic was often given. In 16 such cases with normal rhythm it invariably produced a diuresis ; it never failed even where œdema was absent, though naturally the greatest response was in the œdematous. The previous administration of digitalis appeared to make no difference to the diuretic response, and this confirms the experience of Thomson (1937). Fig. 3 shows the good effect of a mercurial diuretic in four patients who had not been helped by digitalis.

Salyrgan is more than an adjuvant in failure with œdema, but it does not meet the inherent difficulty—myocardial insufficiency. At the best a mercurial diuretic simply removes collected and stagnant fluid and thereby relieves the patient. Digitalis acts often enough under observation, as we have shown, to make it likely that more prolonged use in adequate doses will extend improvement and postpone relapse.

Subsidiary Groups (6–10)

These groups comprising 18 patients (22 tests) with normal rhythm are separately considered for reasons obvious in the diagnosis.

Group 6. *Congenital Heart Disease ; three cases, three tests.*

Two patients, aged 63 and 11, had Fallot's tetralogy. Both responded slightly to digitalis. The remaining patient, aged 43, with coarctation of the aorta, was not improved.

Group 7. *Rheumatic Carditis with Valvular Disease ; seven cases, nine tests.*

The average age was only 17. All had considerable œdema. A single patient improved on digitalis and two others showed a reduction in rate from an average of 125 to 97. Six died later, four within a few months and two within two years.

Group 8. *Infective Endocarditis ; three cases, four tests.*

Post-mortem proof of the diagnosis was obtained in all three cases. In a single patient digitalis increased the urinary output with slight temporary benefit, but no effect at all was demonstrable in the others.

Group 9. *Complete Heart-block ; two cases, two tests.*

Both patients had slight œdema. One improved with a diuresis and a slight fall in ventricular rate from 32 to 27 ; the other was unaffected.

Group 10. *Chronic Nephritis ; three cases, four tests.*

All had cardiac failure. In the one patient without œdema, digitalis relieved the dyspnœa and abolished Cheyne-Stokes respiration and pulsus alternans. The other two patients, with œdema, improved and showed diuresis.

Summary and Conclusions

1. This investigation was undertaken primarily to decide the clinical value of digitalis in heart failure with normal (sinus) rhythm. The secondary purpose was to compare its value in such failure with that in auricular fibrillation, which is better known.

2. Sixty-five patients with heart failure and normal rhythm were observed. After one week or more at rest in bed, digitalis leaf was given in a dose of 2 grains three times a day, i.e. 6 grains (0·4 g.) daily, for one to two weeks. The condition at the beginning of digitalis treatment was compared with that at the end. The criteria of failure were dyspnœa, liver enlargement, and œdema with or without hydrothorax ; and judgment as to improvement was made on these criteria together with diuresis. Reduced heart-rate was not reckoned as a criterion, and it is separately considered. Twenty-five patients had hypertensive heart disease, 12 with and 13 without past coronary thrombosis ; 11 had chronic rheumatic valvular disease ; 9 had syphilitic heart disease ; and 2 were unclassified. In these, the main groups, the majority, 72 per cent., had œdema at the end of the week's rest in bed and a quarter of these had hydrothorax, confirmed by X-ray examination.

3. The control lay in the preliminary rest in bed without digitalis ; but for comparison another series of 30 patients with failure and auricular fibrillation was observed in the same way.

4. Under rest in bed only, in normal rhythm, 17 of 47 (36%) improved ; in auricular fibrillation, 11 of 28 (39%) improved.

5. The main digitalis results are shown in Table I and there are illustrative charts of effect and of failure of effect. In normal rhythm some clinical improve-

ment was demonstrated in 35 out of 58 tests (60%) ; and 23 of 58 showed none. Admittedly 17 received only slight benefit, so that moderate or great benefit resulted in 18 only, i.e. 31 per cent. of the whole. There was little difference in response among the separate ætiological groups, though the rheumatic group responded best (Table I).

The heart-rate in failure with normal rhythm is moderate, the average in our series before digitalis being 85. It was reduced by digitalis in 27 out of the 58 tests (47 patients), i.e. in almost half (46·5%). The average fall in rate among those in whom it occurred was from 85 to 67, i.e. 18. This was usually secured without toxic symptoms, and when these appeared (20%) they were slight (nausea and rarely vomiting). Whatever the initial rate, digitalis often reduced it. Reduction in rate was not always accompanied by clinical improvement, though improvement was rather more common in the patient who showed it. Some good clinical results were seen without any reduction in rate (Table II).

Diuresis was induced by digitalis in 27 of 58 tests (47 patients) in normal rhythm. It was freer in patients with gross œdema than in those with little or none ; but some diuresis was obtained almost as frequently in those without as in those with œdema.

The course of the disease after the onset of failure in normal rhythm is short—18 of 29 patients died within a year—and this in general must lessen the likelihood of improvement from digitalis. Yet in a particular patient the response to digitalis is no guide to the expectation of life ; and if digitalis fails at a first trial, it may occasionally succeed at a later trial.

6. Of the patients with *auricular fibrillation*, 12 had hypertensive heart disease, 14 had chronic rheumatic valvular disease, and 4 were unclassified. Clinical improvement was demonstrated in 23 of 32 tests (30 patients), i.e. in 72 per cent. ; and 9 showed none. It was slight in nine, and moderate or great in fourteen, i.e. 44 per cent. of the whole.

The heart-rate was higher than in the normal rhythm series, an average of 98 against 85, a difference of 13. The rate was reduced by digitalis in the great majority, in 28 of 32 tests (87%). The average fall in rate where a fall resulted was from 100 to 70, an average fall of 30. Most of those with slowing also showed benefit, but in the absence of slowing there was none.

The rheumatic group was distinguished by a higher average rate before treatment, by reduction of the rate in all fourteen cases, and by clinical improvement in all but two. Even with a moderate initial rate (below 100), results were better in rheumatic than in non-rheumatic fibrillation, though no better than in rheumatic cases with normal rhythm.

Without the rheumatic group, the fibrillation series responded to digitalis no better than the normal rhythm series. In hypertension, for instance, the results were similar.

7. A mercurial diuretic sometimes administered after a digitalis test nearly always produced a free diuresis even where digitalis had failed. In œdematous patients other than those with rheumatic auricular fibrillation and a high ventricular rate, a mercurial diuretic usually has more value than digitalis. Yet a trial of digitalis cannot be omitted, for it alone acts directly on the heart.

The partnership of a mercurial diuretic with digitalis should govern the treatment of heart failure.

8. Digitalis is always indicated in congestive heart failure irrespective of rhythm, but it is often inefficient, as it fails completely in about a third of all cases.

In heart failure with normal rhythm digitalis is helpful in rather more than half the cases.

In heart failure with auricular fibrillation, digitalis is more often helpful than it is in normal rhythm, for it benefits more than two-thirds. This superiority rests on its particular value in rheumatic heart failure with fibrillation, largely but not entirely due to the high ventricular rate. Incidentally, it is in this group that rate control by continued digitalis best prevents recurrence of the failure.

The real difference in the response of heart failure to digitalis lies not between auricular fibrillation and normal rhythm, but rather between rheumatic auricular fibrillation and all other kinds of heart failure irrespective of rhythm.

We are indebted to Drs. C. Hollins, R. W. Parnell, and F. S. Cosh, house physicians to the Cardiac Department, for their great assistance in recording clinical observations ; and to the Sisters of the Wards for their valued co-operation. Dr. John Grimshaw has kindly advised us on the text.

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PRÆCORDIAL LEADS IN ELECTRO-CARDIOGRAPHY

From the early days of electrocardiography, attempts to understand the cause of the various waves have been made by the use of leads on different parts of the surface of the heart. This method has not been available for use in man, and clinical study has been mainly confined to the three standard leads, the appearance of which has now become familiar to all. More recently leads from various points over the chest wall have been used and many valuable papers have been published. Unfortunately different conventions have been adopted for placing the electrodes so that comparisons have been difficult, and in some cases the normal curves have shown inverted T waves and in others upright T waves, with corresponding changes in the other complexes. It is not yet certain if the fourth lead, or still other leads, will become essential in routine clinical work, but it is certain that they help greatly in understanding what is taking place in the heart. It is, therefore, important that all these should be as easy to compare as possible and intelligible to as wide a circle as possible.

At the first General Meeting of the Cardiac Society of Great Britain and Ireland, held at Edinburgh on April 15, 1937, Dr. John Cowan proposed that a committee should be appointed to see if it was possible to reach agreement on the points to be adopted for a standard lead IV. This was approved, and fortunately the American Heart Association had already appointed a committee for the same purpose. It proved possible for the two to reach agreed conclusions, embodied in the following memorandum. This was published in the *British Medical Journal* and in the *Lancet* on January 22, 1938, and at the same time in America ; but it seems desirable to reprint it in this Journal, partly for convenience and partly as a record of the work of the first committee appointed by the Cardiac Society.

A JOINT MEMORANDUM

At the request of the Council of the Cardiac Society of Great Britain and Ireland we print below a Memorandum on Prækordial Leads in Electrocardiography. Many different positions have been used for the electrodes in obtaining these electrocardiograms, and much confusion has arisen from the different methods in use. As it has been possible for the Cardiac Society of Great Britain and Ireland and the American Heart Association to reach agreement it is hoped that these joint recommendations will be useful, both to those working at the subject and to all others who are interested.

Recommendations for Standardization

In the last few years electrocardiographic leads in which an electrode placed upon the præcordia is paired with an electrode in contact with some part of the body distant from the heart have come into widespread use. The confusion which has resulted from the lack of uniformity and precision in the technique and nomenclature employed by different observers in connexion with leads of this kind has led to an almost universal desire that a standard practice be established. To this end the Cardiac Society of Great Britain and Ireland and the American Heart Association have each appointed a committee to consider this matter and make recommendations. The two committees have conferred and have agreed jointly to make recommendations with reference to the routine use of a single præcordial lead. It is understood that either committee may make additional reports with reference to multiple præcordial leads and other matters not dealt with in the present report.

1. It is recommended that those who employ a single præcordial lead place the præcordial electrode upon the extreme outer border of the apex beat, as determined by palpation. If the apex beat cannot be located satisfactorily by palpation the electrode may be placed in the fifth intercostal space just outside the left border of cardiac dullness, or just outside the left mid-clavicular line if percussion of the heart is unsatisfactory. Where præcordial leads are taken by a technical assistant, the position for the præcordial electrode should be marked on the chest by the physician.

2. It is recommended that a single præcordial lead in which the præcordial electrode has the location specified in the preceding paragraph be known as lead IV B when this electrode is paired with an electrode in the left interscapular region ; lead IV R when it is paired with an electrode on the right arm ; lead IV L when it is paired with an electrode on the left arm ; lead IV F when it is paired with an electrode on the left leg ; and lead IV T when it is paired with a central terminal connected through equal resistances of 5,000 or more ohms to electrodes on each of the three extremities mentioned.

It is suggested that for all ordinary purposes lead IV R or lead IV F be employed. The latter lead should have the preference until it has been established that the former, which is somewhat more convenient, is equivalent to the latter for all practical purposes, or yields results of equal value.

3. It is recommended that in taking the præcordial leads specified the galvanometer connexions be made in such a way that relative positivity of the apical electrode is represented in the finished curve by an upward deflection (a deflection above the iso-potential level) and relative negativity of the apical electrode by a downward deflection. It is urged that this convention be adhered to in the case of præcordial leads other than those specified, and also in the case of all leads in which one electrode is placed much closer to the heart than the other. In other words, it shall be the standard convention in taking such leads to make the galvanometer connexions in such a way that relative positivity of the electrode nearer the heart is represented by an upward deflection.

4. It is recommended that with the galvanometer connexions made as described in the preceding paragraph the deflections of præcordial leads be designated by the symbols P, Q, R, S, and T, and that in the application of these symbols the same conventions be employed as in the case of the standard limb leads.

5. It is recommended that in taking præcordial leads the electrocardiograph be so adjusted that a deflection of 1 cm. in the finished record corresponds to a potential difference of one millivolt, as in the case of the standard limb leads. Any reduction in sensitivity made necessary by very large deflections should be clearly indicated on the curve, preferably by photographing the effect of introducing a potential difference of 1 mv. into the galvanometer circuit.

6. It is recommended that the greatest dimension of the apical electrode employed in taking the leads specified in this report be 3 cm. or less. A circular electrode between 2 cm. and 3 cm. in diameter should ordinarily be employed.

7. It is recommended that the terms lead IV (R, F, etc.), apical lead, apex-leg lead, etc., be used henceforth only in connexion with the leads specified in this report.

The above recommendations have been drawn up by the following two committees, working in co-operation :

Committee of the Cardiac Society of Great Britain and Ireland

D. Evan Bedford (London)
John Cowan (Glasgow)
A. N. Drury (Cambridge)
I. G. W. Hill (Edinburgh)
John Parkinson (London)
P. H. Wood (London)

Committee of the American Heart Association

Arlie R. Barnes (Rochester)
Harold E. B. Pardee (New York)
Paul D. White (Boston)
Frank N. Wilson (Ann Arbor)
Charles C. Wolferth (Philadelphia)

These joint recommendations have been approved by the Council of the Cardiac Society and by the American Heart Association, who now authorize their publication. The following addendum to the joint report has been made by the Cardiac Society.

Addendum by Cardiac Society

1. The above report deals with the nomenclature and technique for obtaining a single præcordial lead suitable for routine clinical work. The committee of the Cardiac Society had insufficient evidence available to permit their making recommendations in respect of multiple præcordial leads. The American

Heart Association will, however, draw up such recommendations, and, when published, copies of these will be available for those specially interested on application to the secretary of the Cardiac Society (Dr. Maurice Campbell, 25, Upper Wimpole Street, London, W.1).

2. In using either of the two standard præcordial leads—for example, lead IV R or lead IV F—the correct polarity (paragraph 3) is obtained as follows. The lead switch is turned to lead I. The L.A. terminal is connected to the præcordial electrode. The R.A. terminal is connected to the distant electrode—for example, to the right arm for lead IV R, or to the left leg for lead IV F.

CHEST LEADS IN CLINICAL ELECTRO-CARDIOGRAPHY

BY

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In a previous communication Sorsky and Wood (1937) presented the normal appearances of chest lead electrocardiograms, paying particular attention to normal variations and to the position of the proximal electrode. As the question of standardizing the technique for chest leads was then under consideration by the Cardiac Society of Great Britain and Ireland and the American Heart Association, further work was held up pending their conclusions, which have now been published (1938). On the question of polarity it was agreed that the galvanometer connections should be so arranged that relative positivity of the proximal electrode yields an upward deflection on the electrocardiogram. By lead IV is meant a chest lead in which the proximal electrode is placed upon the apex beat. When this is paired with the right arm it is called lead IV R, and when it is paired with the left leg it is called lead IV F. In all our work we have connected the proximal electrode to the left arm terminal, so that lead IV R is obtained by setting the lead selector dial on the electrocardiograph to lead I, and lead IV F by setting this dial to lead III, after interchanging the terminals in order to correct the polarity.

In our previous work on 150 normal controls, our description of the normal variations of the chest lead electrocardiogram was in terms of the opposite polarity to that now accepted, and based on chest leads paired with the left leg. It is now necessary to refer to our records of chest leads paired with the right arm, in which the accepted polarity was obtained. From an analysis of these electrocardiograms we may present the normal appearances of lead IV R as follows :

1. The P wave is upright and averages 0·14 mv. in amplitude, with a maximum of 0·25 mv.

2. A Q wave is present in 22 per cent. and averages 0·15 mv. in amplitude with a maximum of 0·5 mv.

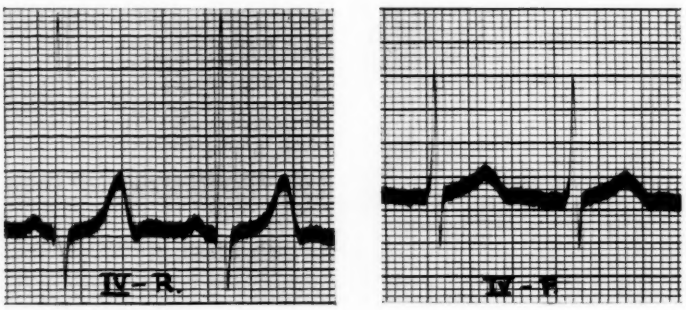
3. The main QRS deflection is upright and is never monophasic. R is very tall and S averages 0·75 mv.

4. The RS-T segment usually lies at the iso-potential level, but it may be elevated to a maximum of 0·1 mv., or rarely it may be slightly depressed.

† Received August 26, 1938.

5. The T wave is always upright and averages 0·7 mv. in amplitude, ranging between 0·3 and 1·4 mv.

Lead IV F is similar except that the P wave is usually diphasic or inverted and is often very small. Typical records are shown in Fig. 1 (A and B).



A B

FIG. 1.—Normal chest-lead electrocardiograms. (A) Lead IV R. (B) Lead IV F.

In addition to leads IV R and F we have studied four other chest leads : (1) the left pectoral—right arm lead ; (2) the left pectoral—left leg lead ; (3) the right pectoral—right arm lead ; (4) the right pectoral—left leg lead. In the right pectoral leads the proximal electrode was placed in the fourth intercostal space at the right border of the sternum ; in the left pectoral leads it was placed midway between this point and the apex beat * (see Fig. 2).

In normal controls it was found that in the apical leads the R deflection dominated the QRS complex, but that shifting the proximal electrode to the right favoured the S deflection, so that in the left pectoral leads R and S were more or less of equal amplitude, and in the right pectoral leads S was the dominant deflection. This phenomenon is illustrated in Fig. 3A, and was found in 75 per cent. of the controls when the proximal electrode was paired with the right arm, and in 95 per cent. when the proximal electrode was paired with the left leg.

The effect upon the T wave of shifting the proximal electrode to the right differed according to the distal electrode employed. When the right arm was used the T wave remained upright even in the right pectoral lead. But when the left leg was used the T wave often became inverted. Thus in the left pectoral—left leg lead the T wave was inverted in 21 per cent. of normal children and occasionally in slim young adults, and in the right pectoral—left leg lead it was inverted in 65 per cent. of normal children and in 58 per cent. of normal adults (see Table I). An electrocardiogram showing inversion of the T waves in the left and right pectoral—left leg leads in a normal child is shown in Fig. 3B.

* In the terminology recommended by the American Heart Association, our right pectoral —right arm lead would be called lead CR 1, and our left pectoral—right arm lead, though it has no exact counterpart, corresponds roughly to lead CR 3.

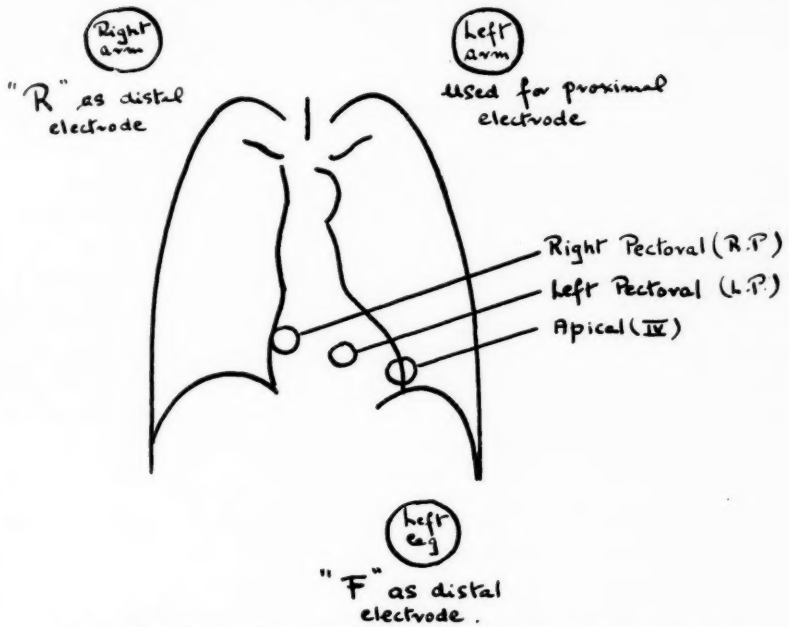
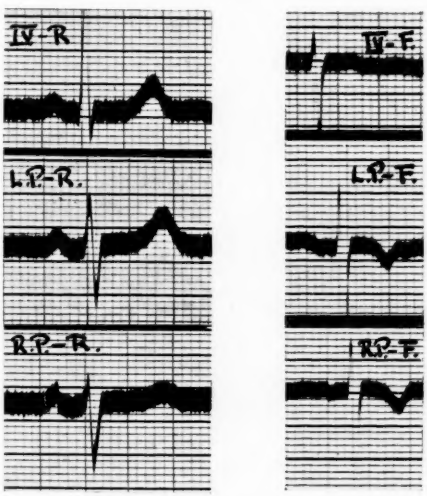


FIG. 2.—Diagram showing positions of the proximal electrode in relation to the underlying heart.



A B

FIG. 3.—Normal chest leads showing the behaviour of the QRS complex (A), and of the T wave in a child (B), as the proximal electrode is shifted from the apical (top) to the left pectoral (middle) and to the right pectoral (bottom) position.

TABLE I

INVERSION of the T WAVES in PECTORAL LEADS PAIRED WITH THE LEFT LEG

	LEADS		
	TV - F	L.P. - F.	R.P. - F
CHILDREN	RARE	21 %	65 %
ADULTS	NIL	RARE	58 %

The present report deals with the chest lead electrocardiograms obtained from 314 patients selected for different purposes. No case was included in which the diagnosis was in doubt. In the majority nine tracings were taken, three with limb leads and six with chest leads. Serial records were obtained in about a third of the cases, particularly in those which we thought might show transient changes. Many graphs were discarded because of uncertainty of diagnosis or because of mixed etiological factors. The total number of chest lead tracings taken has been estimated at 3,000, excluding normal controls. The object of the work was twofold : to determine the best distal electrode ; and to assess the value of multiple chest leads. This necessitated a wide investigation into the value of chest leads in general and in all types of heart disease. The material is analysed in Table II.

TABLE II

MATERIAL

314 SELECTED CASES

ACTIVE RHEUMATIC CARDITIS	30
PERICARDITIS	7
LEFT VENTRICULAR ENLARGEMENT	120
CARDIAC DISPLACEMENT	12
RIGHT VENTRICULAR ENLARGEMENT	50
BUNDLE BRANCH BLOCK	20
DIGITALIS EFFECT	15
MYOCARDIAL INFARCTION	33
ANGINA PECTORIS	27

Cases of thyrotoxicosis, bacterial endocarditis, and of congenital heart disease without cardiac enlargement produced no special electrocardiographic changes with limb or with chest leads, and have not been included in this series.

Cases of aortic valvular disease of any etiology have been grouped with cases of hypertensive heart disease under the heading of left ventricular enlargement if that was present, and cases of mitral stenosis, pulmonary stenosis, and cor pulmonale have been grouped together under the heading of right ventricular enlargement unless there was evidence to the contrary. We also studied thirty cases of severe anæmia, five of myxoedema, and one case of toxic myocarditis from pneumonia. Chest leads proved of no help in the diagnosis of these conditions, the changes in both limb leads and chest leads being of a similar order.

Rheumatic Carditis

Electrocardiographic evidence of rheumatic carditis is more or less confined to prolongation of the P-R interval, to nodal rhythm, heart block, and to bundle branch block, i.e. to conductive defects. Alterations in the RS-T segment or in the T wave in limb lead electrocardiograms are usually due to pericarditis. It has been stated, however, that changes in the T wave may occur with chest leads. Thus Levy and Bruenn (1935) found frequent alterations in the direction and voltage of the T wave in serial chest lead records in cases of rheumatic carditis. Their results have already been criticized (Sorsky and Wood, 1937) on the grounds that normal children frequently show reversal of the T wave in the lead which they used. Robinow, Katz, and Bohning (1936) made a more careful study of cases of rheumatic carditis, checking their findings against a group of normal controls, and they concluded that reversal of the T wave in chest leads was more common in children with rheumatic carditis than in normal children.

It must be remembered that reversal of the T wave is found in the left pectoral—left leg lead in 21 per cent. of normal children, and that the T wave is diphasic in this lead in another 18 per cent. (Sorsky and Wood, 1937). We wish to emphasize the fact that such reversal of the T wave rarely occurs if the proximal electrode is paired with the right arm, or if it is placed on the apex beat and not within it.

Bearing these facts in mind we have analysed the chest-lead electrocardiograms of thirty cases of active rheumatic carditis, excluding those with pericarditis. In many serial records were obtained.

The QRS complex was always normal. The RS-T segment did not deviate from the iso-potential level. There were, however, changes in the direction of the T wave in some instances. When the proximal electrode was paired with the right arm the T wave was inverted in three cases in the apical lead, in three cases in the left pectoral lead, and in ten cases in the right pectoral lead ; when the proximal electrode was paired with the left leg the T wave was inverted in five instances in the apical lead, in ten instances in the left pectoral lead, and in twenty in the right pectoral lead. These figures are expressed per cent. in Table III. If the T wave was inverted in the apical or in the left pectoral lead, it was also inverted, and usually more so, when the proximal electrode was shifted to the right. This rule is always obeyed by normal controls, and it will

be shown later that it also holds good for cases with right ventricular stress or enlargement. The above figures show that the T wave is more often inverted in children with rheumatic carditis than in normal children. When the proximal electrode is paired with the right arm inversion of the T wave is more significant though less common than when it is paired with the left leg.

It is concluded that chest leads may be of value as an aid in the diagnosis of active rheumatic carditis ; that this value is enhanced if the proximal electrode is paired with the right arm, and is reduced if it is paired with the left leg ; that the changes consist of inversion of the T wave, and occur least in the apical lead and most frequently in the right pectoral lead ; that if there is inversion of the T wave in any lead there is usually less inversion if the proximal electrode is shifted to the left and more inversion if it is shifted to the right ; and finally that, as will be shown subsequently, these changes are similar to those produced by stress or enlargement of the right ventricle from any cause, so that it is possible that the changes of rheumatic carditis merely represent right ventricular stress.

TABLE III

INVERSION of the T WAVE IN CASES of RHEUMATIC CARDITIS

CHEST LEAD	R.	F.
IV	10 %	16·6 %
LEFT PECTORAL	10 %	33·3 %
RIGHT PECTORAL	33·3 %	66·6 %

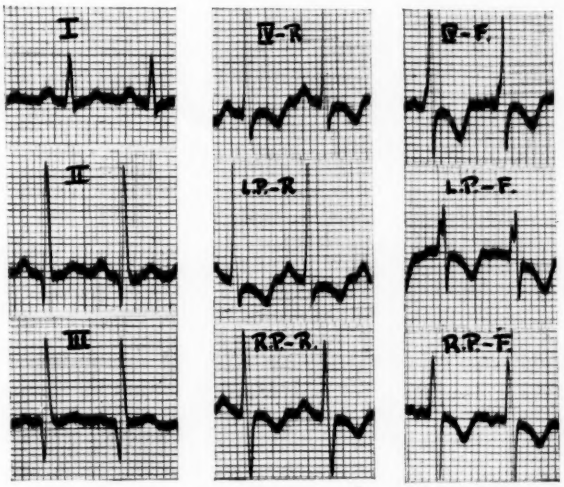
PERICARDITIS

Wood (1937) has recently described the changes in limb lead electrocardiograms in cases of pericarditis as being of a T_2 pattern, both the early elevation of the RS-T segment and the later inversion of the T wave being maximum in lead II.

Vander Veer and Norris (1937) showed illustrations of chest lead tracings in four cases of pericarditis which exhibited typical changes of a T_2 pattern in serial limb leads. For some reason these chest lead tracings were quite normal, and the authors concluded that the electrocardiographic changes of pericarditis could be distinguished from those of myocardial infarction by the fact that the former did not occur in chest leads. At the time of their publication we knew that this conclusion was incorrect, and since then Bellet and McMillan (1938) have reported marked changes in chest leads in cases of pericarditis. Using the old polarity they found initial depression of the RS-T segment and later reversal of the T wave. These changes often lasted longer than those in the limb leads. In some cases they found that the abnormality in the chest lead

only appeared when the proximal electrode was placed over the area of pericardial friction.

We used chest leads in seven cases of pericarditis, six of which exhibited limb lead changes of the T_2 pattern. Two were rheumatic, two malignant, one tuberculous, one infective, and one was a case of chronic constrictive pericarditis. An effusion was present in five of them. Only one showed elevation of the RS-T segment in the early phase in limb leads, and this elevation was more evident in the chest leads. Inversion of the T wave occurred in all cases in the apical and in the left pectoral leads, and usually in the right pectoral leads as well, and were equally evident with either distal electrode. Of the seven cases, two came to autopsy and two were chronic, so that the changes during the period of recovery were only studied in three ; limb leads were normal in one, and inverted T waves persisted longer in chest than in limb leads in the other two. The degree of T wave inversion was greater in chest leads, and the form of the QRS complex was always normal. These facts are illustrated in Figs. 4 and 5.



A B C

FIG. 4.—Electrocardiograms from a case of rheumatic pericarditis with effusion, showing normal limb leads (A), and inversion of the T wave in all chest leads whether paired with the right arm (B), or with the left leg (C).

It is concluded that in cases of pericarditis changes in the RS-T segment and in the T wave are well shown in chest leads ; that these changes may last longer than the changes in limb leads, and that they may occur in association with normal limb leads. Chest leads may therefore be of value. Multiple leads may be useful to distinguish pericarditis from rheumatic carditis, for in the former the right pectoral lead may be normal, whereas in the latter if the T wave is inverted in the apical or in the left pectoral lead it will also be

inverted in the right pectoral lead. To distinguish pericarditis from anterior myocardial infarction attention should be paid to the QRS complex rather than to the T wave. For the distal electrode we prefer the right arm in children, but the left leg is equally suitable in adults.

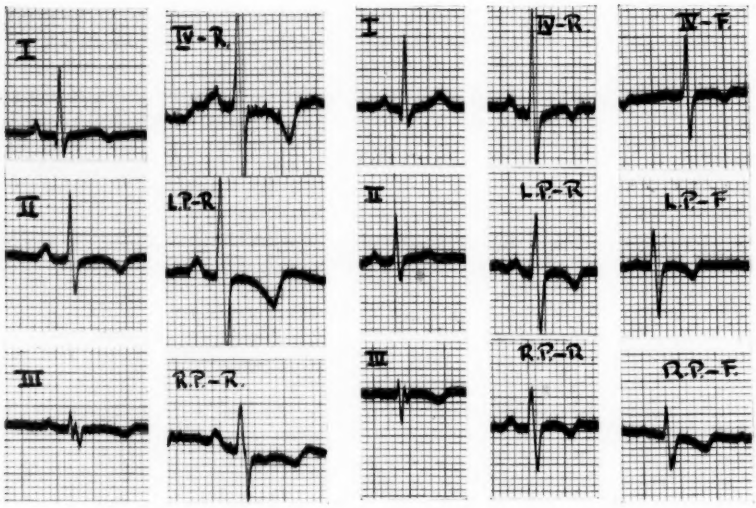


FIG. 5.—Electrocardiograms from a case of rheumatic pericarditis without effusion, showing how changes in chest leads may outlast those in limb leads.

(A) Limb leads showing a typical T_2 pattern.
(B) Chest leads paired with the right arm (same date).
(C) Limb leads ten weeks later showing normal T waves.
(D) Chest leads paired with the right arm (same date as C).
(E) Chest leads paired with the left leg (same date as C).

LEFT VENTRICULAR ENLARGEMENT

There have been but few reports on the appearance of chest lead electrocardiograms in cases of left ventricular enlargement. Van Nieuwenhuizen and Hartog (1937b), using a left pectorial—left leg lead, found that there was a decrease in voltage of the R wave and an increase in voltage of the S wave in cases of hypertensive heart disease, so that R often measured less than 2 mm. and S more than 17 mm. In some cases they found absence of the R wave altogether. (To avoid confusion their terminology has been translated into the current one). Their left pectoral lead was taken from a fixed point on the chest wall close to the left border of the sternum, and was therefore very far to the right of the apex beat in cases with considerable left ventricular enlargement.

Hecht (1936), employing Wilson's neutral electrode (Wilson and others, 1934d), considered that inversion of the T wave in the apical lead in cases with

enlargement of the left ventricle indicated serious damage to the myocardium. His proximal electrode was placed in the anterior axillary line.

Roth (1937), using the old polarity, found reversal of the T wave in lead IV F, but not in the left pectoral—left leg lead, in some cases of hypertensive heart disease in which the apex beat was considerably displaced to the left. He concluded that lead IV F was simply lead III in such cases, and that the left pectoral lead was therefore the better chest lead. We find this argument a little difficult to follow. If the proximal electrode is placed over a heaving cardiac impulse in the anterior axillary line or thereabouts, the electrocardiogram so obtained cannot be very different from one obtained by a direct lead from the surface of the apex of the left ventricle (Wilson and others, 1932 b). Considering the position of the left ventricle in these cases it is likely that similar changes of potential will be found in the left arm, which because of its proximity will dominate the limb leads. In short, in such cases, lead III (and lead I) are almost semi-direct leads, the left arm acting almost as a proximal electrode. We fail to see that this detracts from the importance of lead IV, except that by examining the limb leads we might predict the appearance of lead IV.

Holzmann (1937), using an anterior-posterior lead and the new polarity. showed many examples of very small R waves and deep S waves in cases with considerable enlargement of the left ventricle. The T wave remained upright, for the proximal electrode was close to the sternum.

We have studied the chest lead electrocardiograms in 120 cases of left ventricular enlargement. The behaviour of the QRS complex was interesting. In the apical leads R was usually very tall, but in the left pectoral leads it often measured less than 2 mm. in height, and in the right pectoral leads it was even less conspicuous. In the right pectoral—right arm lead S was the dominant deflection in 97 per cent. of the cases compared with the 75 per cent. figure for normal controls. A monophasic downward deflection was rare in the left pectoral leads, occurring in two cases of syphilitic aortic incompetence and in one case of hypertensive heart disease, but in the right pectoral leads it occurred in three cases of syphilitic aortic incompetence and in four cases of hypertensive heart disease. There was no evidence of myocardial infarction in any of these cases.

Our findings with regard to the QRS complex are therefore in agreement with those of Van Nieuwenhuizen and Hartog (1937b), and with those of Holzmann (1937), and we conclude that very small R waves in the left or right pectoral leads cannot be accepted as evidence of myocardial infarction in cases with left ventricular enlargement, and that, in the presence of the latter, even monophasic downward deflections in the left pectoral lead must be interpreted with caution.

In studying the behaviour of the RS-T segment and of the T wave, we divided the cases into three groups according to the degree of enlargement of the left ventricle. This was always estimated radiologically by the same observer, P. W., throughout this work.

Group I consisted of fifty cases of slight left ventricular enlargement, most from hypertension, a few from aortic valvular disease. In some there was mild

to moderate breathlessness on exertion, and in others there were no cardiac symptoms. Limb leads showed left axis deviation with an upright T wave in lead I. Chest leads paired with the right arm yielded normal curves in every instance, wherever the proximal electrode. Chest leads paired with the left leg, however, showed a decrease in the incidence of an inverted T wave in the right pectoral lead from 58 per cent. in the controls to 14 per cent.

Group II consisted of fifteen cases with moderate left ventricular enlargement, mostly from hypertension. Effort was more limited by dyspnœa than in the previous group, but there was no orthopnœa or other evidence of left ventricular failure. Limb lead electrocardiograms showed left axis deviation with an upright T wave in lead I, some with and some without depression of the RS-T segment in that lead. In the former cases leads IV R and F showed a similar depression of the RS-T segment. Otherwise, and apart from the QRS changes previously mentioned, chest leads paired with the right arm were normal, but chest leads paired with the left leg showed an even lower incidence (one case only) of inversion of the T wave in the right pectoral lead than occurred in group I.

In group III there were fifty-five cases with considerable left ventricular enlargement, thirty-nine from hypertension, fifteen from aortic incompetence (mostly syphilitic), and one from aortic stenosis. Most had some evidence of left ventricular failure, and some had right ventricular failure also. Cases with angina pectoris or with a history of myocardial infarction were excluded. With limb leads there was inversion of the T wave in lead I only in thirty-three instances, in leads I and II but not in lead III in eleven instances, and in all three leads in five. In nearly all of these there was associated depression of the RS-T segment in the lead or leads in which the T wave was inverted. Depression of the RS-T segment in lead I without inversion of the T wave occurred in five of the remaining six cases, and a flat T wave in lead I was found in the sixth. Left axis deviation was always present except in those cases with inversion of the T wave in all leads.

With chest leads there was inversion of the T wave with associated depression of the RS-T segment in the apical leads alone in twenty-five instances, in both the apical and left pectoral leads but not in the right pectoral leads in three instances, and in all leads in two. When inversion of the T wave was confined to lead I in the limb leads, it was also confined to leads IV R and F in the chest leads ; when it occurred in leads I and II, it was found in three out of eleven such cases in both the apical and left pectoral leads ; and when it happened in all three limb leads it also happened in two out of five such cases in all three chest leads. These findings are illustrated in Fig. 6. When depression of the RS-T segment in lead I was the only change in limb leads, a similar depression of the RS-T segment was found in leads IV R and F but not in the other chest leads. The majority of cases in group III exhibited slight elevation of the RS-Tsegment in the pectoral leads, and it was this group especially which showed the small R waves and the large S waves previously described. Finally, it should be noted that of the fifty-five cases in this group only two showed inversion of the

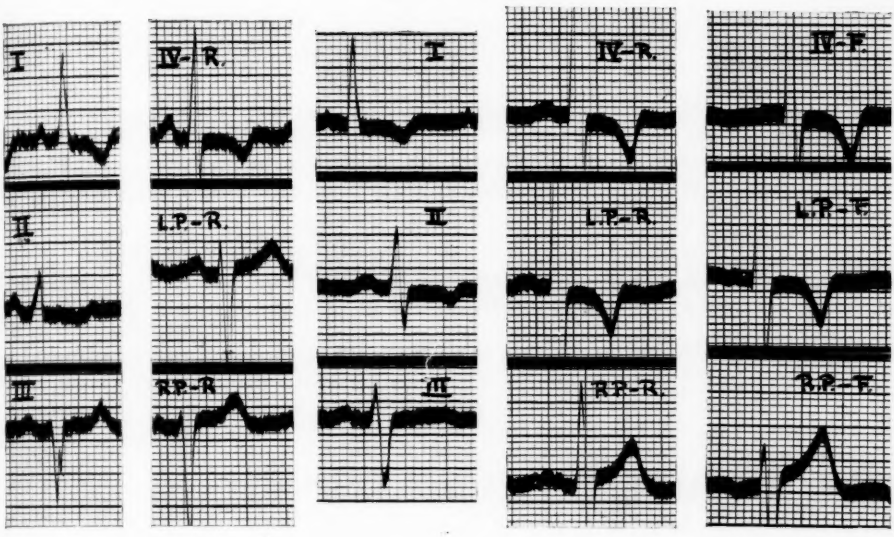


FIG. 6.—Electrocardiograms from two cases of hypertension with considerable enlargement of the left ventricle.

A and B. Limb leads (A), and chest leads paired with the right arm (B), in a case in which inversion of the T wave is confined to leads I and IV.

C, D and E. Limb leads (C), and chest leads paired with the right arm (D), and with the leg (E), in a case in which inversion of the T wave has extended to lead II and to the left pectoral leads in addition to leads I and IV.

T wave in the right pectoral—left leg lead, and in these two the T wave was inverted in every other lead including the three limb leads.

From a consideration of the facts which we have presented certain points arise which merit further attention.

First, the facts summarized in Table IV show that with progressive enlargement of the left ventricle the incidence of inversion of the T wave in the right pectoral—left leg lead fell from the normal figure of 58 per cent. to nil (excluding rare cases in which the T wave was inverted in all leads). In other words, if the left ventricle was significantly enlarged the T wave was upright in the right pectoral—left leg lead ; if the T wave was inverted in this lead, the left ventricle was not significantly enlarged. We wondered whether displaced hearts due to a high diaphragm, which gave rise to left axis deviation in the standard leads, would produce an upright or an inverted T wave in this lead. To determine the answer to this question we studied normal pregnant women just before labour. We found twelve cases with left axis deviation in the limb leads. Of these, ten showed inversion of the T wave in the right pectoral—left leg lead, and two showed a diphasic T wave. In addition the T wave was inverted in four instances in the left pectoral—left leg lead, and in two instances in lead IV F. We conclude that in the right pectoral—left leg lead we have a means of distin-

TABLE IV

LEFT VENTRICULAR ENLARGEMENT

INVERSION OF THE T WAVE IN THE RIGHT PECTORAL—LEFT LEG LEAD

	No. of cases	Percentage with inverted T wave
NORMAL ADULTS	50	58 %
LEFT VENTRICLE +	50	14 %
LEFT VENTRICLE ++	15	7 %
LEFT VENTRICLE +++	55	0 %*

* Excluding two cases with inverted T waves in all leads

guishing enlargement of the left ventricle from displacement of the heart due to a high diaphragm. If the T wave is upright no inference can be drawn, but if it is inverted significant enlargement of the left ventricle is unlikely. We have found this rule of value in attempting to assess the size of the left ventricle in cases of obesity. When the proximal electrode is paired with the right arm the results are far less helpful because the T wave is very rarely inverted in this lead. Electrocardiograms showing the absence of significant left ventricular enlargement in a case of obesity with hypertension are illustrated in Fig. 7.

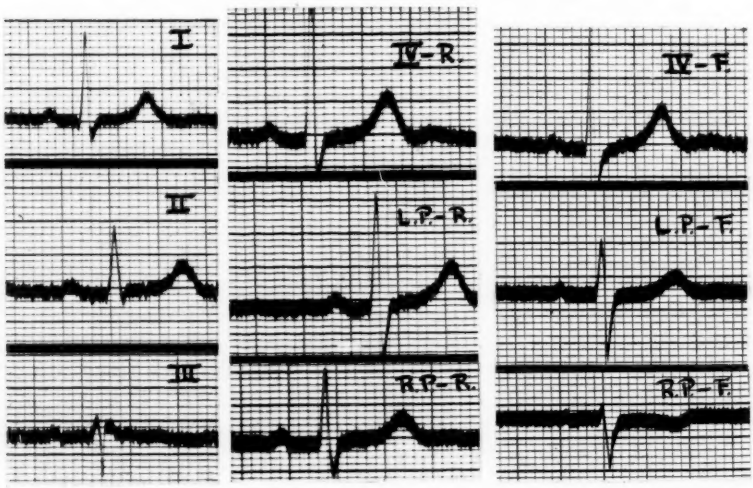


FIG. 7.—Limb leads (A), and chest leads paired with the right arm (B), and with the left leg (C), in a case of obesity. Note the inverted T wave in the right pectoral—left leg lead.

Secondly, the point arises as to why the T wave should be inverted in lead IV in only eighteen out of thirty-three cases of considerable left ventricular enlargement in which it was inverted in lead I. On re-examining these records we found that when the T wave was inverted in lead IV the R deflection dominated the QRS complex, whereas when the T wave was upright the S deflection dominated. This was clear evidence that the proximal electrode had been placed within the apex beat in these latter cases, so that the leads were more left pectoral than apical. We have since re-investigated a few of these patients and have found that great care had to be taken in placing the proximal electrode, but that when this was placed truly upon the apex beat the T wave became inverted and the R deflection dominated the QRS complex as expected.

Thirdly, the facts show that in cases of left ventricular enlargement if the T wave is inverted in any chest lead, it is also inverted (and usually more so) in leads in which the proximal electrode is shifted to the left. This is the reverse of what happens in normal children and in cases of rheumatic carditis, and, as will be shown subsequently, in cases of right ventricular enlargement.

Fourthly, it is evident that depression of the RS-T segment and inversion of the T wave in lead IV R or F do not indicate myocardial infarction in the presence of sufficient enlargement of the left ventricle.

Summary of the effects of left ventricular enlargement on the chest lead electrocardiogram

1. Though the R deflection is often exceedingly tall in leads IV R and F, there is an increased incidence of small R waves and deep S waves in the pectoral leads.

2. With progressive enlargement of the left ventricle the incidence of inversion of the T wave in the right pectoral—left leg lead falls steadily from 58 per cent. in the controls to nil in the cases with considerable enlargement. Patients with cardiac displacement due to a high diaphragm usually exhibit an inverted T wave in this lead.

3. In the lesser degrees of left ventricular enlargement the RS-T segment and the T wave are normal in the apical and left pectoral leads.

4. In the greater degrees of left ventricular enlargement the RS-T segment is depressed and the T wave is inverted in leads IV R and F, and occasionally in the left pectoral leads.

Right Ventricular Enlargement

Groedel (1934) maintains that deformities in the QRS complex and inversion of the T wave in right ventricular semi-direct leads indicate disease of the right ventricle. Hecht (1936), using Wilson's neutral distal electrode, found that right ventricular enlargement caused inversion of the T wave in the right pectoral lead, and sometimes a large Q wave.

To determine the effect of right ventricular enlargement upon the chest

lead curves we investigated thirty-five cases of mitral stenosis, six of chronic cor pulmonale, five of acute cor pulmonale following pulmonary embolism, and four of congenital pulmonary stenosis, making a total of fifty cases with right ventricular enlargement or stress. Patients with aortic valvular disease or with hypertension in addition were excluded.

The QRS complex was usually normal in the apical and in the left pectoral leads, but in the right pectoral leads the main QRS deflection was upright in about a third of the cases, rather more than a third when the proximal electrode was paired with the right arm, and rather less than a third when it was paired with the left leg. Pronounced notching of the R wave was sometimes observed associated with delay in the onset of the intrinsic downward deflection, and occasionally there was prominent Q wave, both these abnormalities being confined to the right pectoral leads. These features are illustrated in Fig. 8.

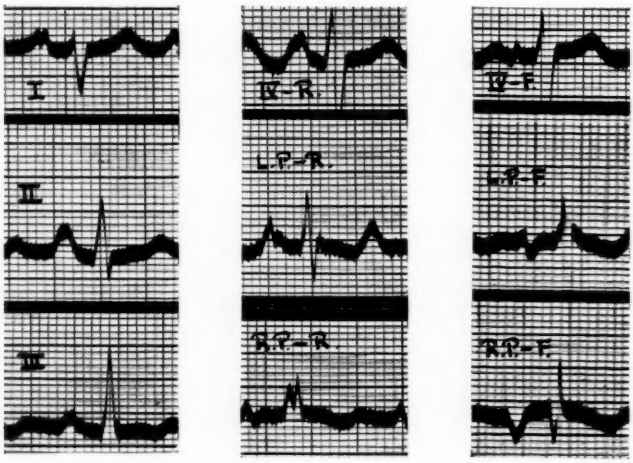


FIG. 8.—Limb leads (A), and chest leads paired with the right arm (B), and with the left leg (C), in a case of mitral stenosis with enlargement of the right ventricle. Note the absence of S waves and the inverted T waves in the right pectoral leads, and the Q wave in the right pectoral—left leg lead.

The frequency of inversion of the T wave in leads paired with the left leg is shown in Table V. (The incidence was somewhat lower in leads paired with the right arm.) It was found in 20 per cent. in lead IV, in 35 per cent. in the left pectoral lead, and in 75 per cent. in the right pectoral lead. As in normal children and as in cases of rheumatic carditis, if the T wave was inverted in any lead it was also inverted, and usually more so, when the proximal electrode was shifted to the right. In table V the incidence of inversion of the T wave in right ventricular enlargement is compared with that in normal adults, in normal children and in rheumatic carditis. This comparison suggests that the cause of inversion of the T wave in normal children and in rheumatic carditis is relative

TABLE V

RIGHT VENTRICULAR ENLARGEMENT

INVERSION of the T WAVE in LEFT LEG paired LEADS

	— LEAD —		
	IV. F.	L.P. – F.	R.P. – F.
NORMAL ADULTS	0 %	2 %	58 %
NORMAL CHILDREN	0 %	21 %	65 %
RHEUMATIC CARDITIS	16·6 %	33·3 %	66·6 %
R.V. ENLARGEMENT	20 %	35 %	75 %

right ventricular dominance, and this finds support in the fact that increased left ventricular dominance has an opposite effect upon the T wave. It will be remembered that when the left ventricle is considerably enlarged the T wave is never inverted in the right pectoral—left leg lead (except in rare cases in which T is inverted in all leads including the three limb leads). With moderate left ventricular enlargement the T wave is inverted in 7 per cent. in this lead ; with slight left ventricular enlargement it is inverted in 14 per cent. ; in normal adults in 58 per cent. ; in normal children in 65 per cent. ; in cases of rheumatic carditis in 66·6 per cent. ; and finally in cases of right ventricular enlargement in 75 per cent. We have expressed these facts altogether in Table VI. This shows clearly the influence of right ventricular enlargement upon the direction of the T wave in the chest lead electrocardiograms employed.

TABLE VI

THE INFLUENCE OF VENTRICULAR ENLARGEMENT UPON THE DIRECTION OF THE T WAVE

Percentage of cases with inversion of the T wave

	Lead IV-F	Lead L.P.-F	Lead R.P.-F.
L.V. ENLARGEMENT +++	58	9	4
L.V. ENLARGEMENT ++	0	0	7
L.V. ENLARGEMENT +	0	0	14
NORMAL ADULTS	0	2	58
NORMAL CHILDREN	0	21	65
RHEUMATIC CARDITIS	16·6	33·3	66·6
R.V. ENLARGEMENT	20	35	75

In general, the extent of T wave inversion was parallel to the degree of right ventricular enlargement, and when the T wave was inverted in lead IV the right ventricle was usually considerably enlarged. Illustrations of inverted T waves in cases of right ventricular enlargement are shown in Fig. 9.

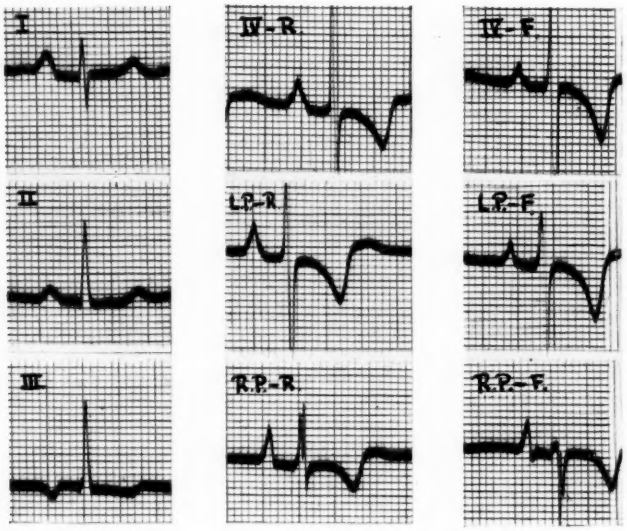
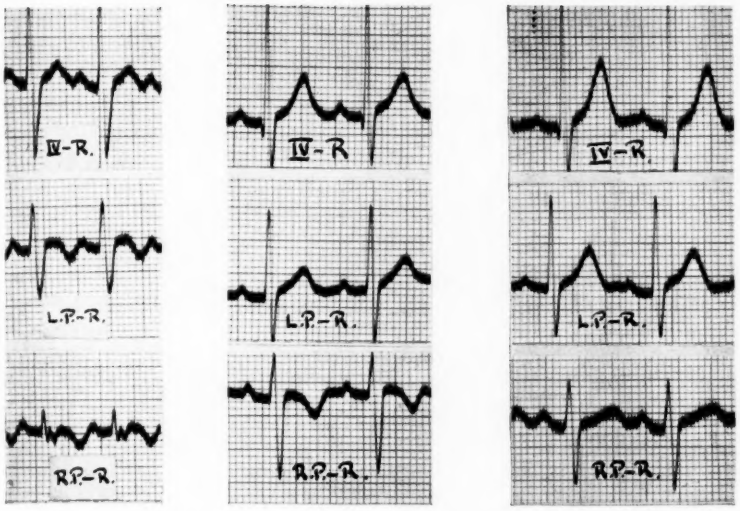


FIG. 9.—From a case of mitral stenosis with considerable enlargement of the right ventricle.
(A) Limb leads.
(B) Chest leads paired with the right arm.
(C) Chest leads paired with the left leg.
Note inversion of the T wave in all chest leads (the tall spiked P wave in the right pectoral leads probably indicates right auricular hypertrophy).

Special attention must be paid to the cases of acute cor pulmonale following pulmonary embolism. In these, inversion of the T wave was transient, and was most marked and lasted longest in the right pectoral leads. The sequence of events was remarkably similar in all five cases. The first record, obtained within twenty-four hours from the onset, showed inversion of the T wave in both pectoral leads and sometimes in the apical leads as well ; the first change occurred two or three days later when the T wave in lead IV became upright if it had been previously inverted ; the second change, after a few more days, occurred when the T wave became upright in the left pectoral lead ; and finally, the T wave became upright in the right pectoral lead. The usual sequence of events is illustrated in Fig. 10.

These findings are important for two reasons : first, because they suggest that inversion of the T wave of the type described may occur from right ventricular stress quite apart from hypertrophy ; and secondly, because they may be of great help in the differential diagnosis of pulmonary embolism from posterior myocardial infarction.



A B C

FIG. 10.—Serial chest lead electrocardiograms paired with the right arm from a case of acute cor pulmonale following pulmonary embolism.

(A) Within twenty-four hours of the onset, showing inversion of the T wave in the left and right pectoral leads.
(B) A week later, showing inversion of the T wave in the right pectoral lead only.
(C) Eighteen days after the onset, showing upright T waves in all leads.

Summary of the effects of right ventricular enlargement upon the chest lead electrocardiogram

1. The main QRS deflection is upright in the right pectoral leads in about a third of the cases. Such curves are especially significant when the proximal electrode is paired with the left leg.

2. Marked notching of the R wave associated with a late intrinsic downward deflection may sometimes occur in the right pectoral lead, and occasionally there may be a prominent Q wave.

3. Inversion of the T wave is common and obeys the rule which may be applied to normal children and to cases of rheumatic carditis, i.e. if the T wave is inverted in any lead, it is also inverted (and usually more so) in leads in which the proximal electrode is shifted to the right.

4. Transient T wave changes of the same type are seen in cases of acute cor pulmonale following pulmonary embolism, and may be of considerable help in diagnosing the condition from posterior basal myocardial infarction.

5. The incidence of inversion of the T wave is greater when the proximal electrode is paired with the left leg ; but its significance is greater when the proximal electrode is paired with the right arm.

These findings are in contrast to those of left ventricular enlargement.

Bundle branch block

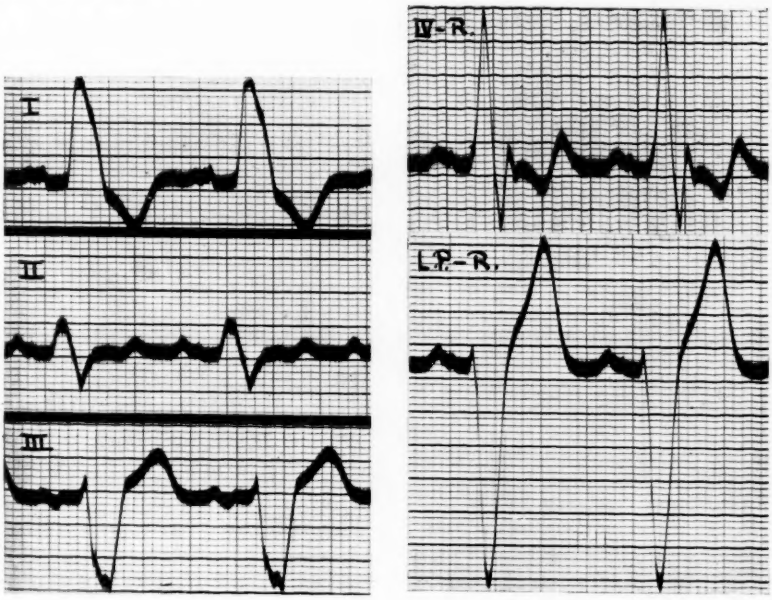
The form of the chest lead electrocardiogram in cases of bundle branch block in human beings was originally worked out by Wilson, Macleod and Barker (1932 b), their publication appearing one month after the original article by Wolferth and Wood (1932 a) recommending the use of chest leads as an aid in the diagnosis of myocardial infarction. Wilson, Macleod and Barker (1932 b) used multiple chest leads paired with the left leg, and these would now be called leads CF 1, 2, 3 and 4 (*Standardization of Præcordial Leads*, 1938). The object of their paper was to show that what was then called right bundle branch block was in reality left bundle branch block and vice versa. Using the old polarity they determined that " when bundle branch block is present the chief upstroke of the ventricular complex in semi-direct leads is early when the exploring electrode is placed close to the ventral surface of the contralateral ventricle and late when this electrode is placed close to the surface of the homolateral ventricle. In the former case the fibre usually remains above, in the latter case it usually remains below the base line throughout the greater part of the QRS interval." They also stated that the right ventricle was favourably situated for semi-direct leads from its ventral surface, but that the left ventricle was not so favourably situated.

Wilson, Johnston, Hill, Macleod and Barker (1934 c), using similar multiple leads but paired with a neutral indifferent electrode, showed that an abnormally long QRS interval and a broad S deflection in lead I signified right bundle branch block. Chest leads V1, V2 and V3 exhibited a late chief upstroke (old polarity) and leads V4 and V5 an early one.

In certain cases of septal infarction with bundle branch block, Wilson, Hill and Johnston (1934 a) advise the use of chest leads for proper interpretation. They described the production of right bundle branch block of the type just mentioned by experimental infarction of the interventricular septum in three dogs. Wilson, Johnston and Barker (1934 b) also advise the use of chest leads in order to interpret limb lead electrocardiograms which exhibit a bizarre form of bundle branch block. In three such cases the limb lead electrocardiograms were illustrated and were thought to be suggestive of left rather than right bundle branch block, but chest leads showed clearly that they signified right bundle branch block.

Our series includes twenty cases of bundle branch block, ten left and ten right. We found that the appearances in the left and right pectoral leads were consistent in all cases. In left bundle branch block (new terminology) the main QRS deflection was downwards with an early intrinsic downward deflection, followed by a large upright T wave ; in right bundle branch block of the type described by Wilson and his associates (1934 c), and by Evans and Turnbull (1937) in this country, the main QRS deflection was upwards with a late intrinsic downward deflection and the T wave was inverted. The appearances in the apical leads were less consistent, no doubt depending upon whether the proximal electrode represented a left or a right ventricular or a septal contact. Thus in left bundle branch block seven cases presented similar features to those in the

pectoral leads, and only three showed a late intrinsic downward deflection with an upright QRS complex and inversion of the T wave. In right bundle branch block the intrinsic downward deflection was early in lead IV in seven cases, followed by a broad sluggardly S deflection and an upright T wave, but in the other three cases the appearances were similar to those in the pectoral leads (see Figs. 11 and 12).



A B

FIG. 11.—Left bundle branch block (new terminology).

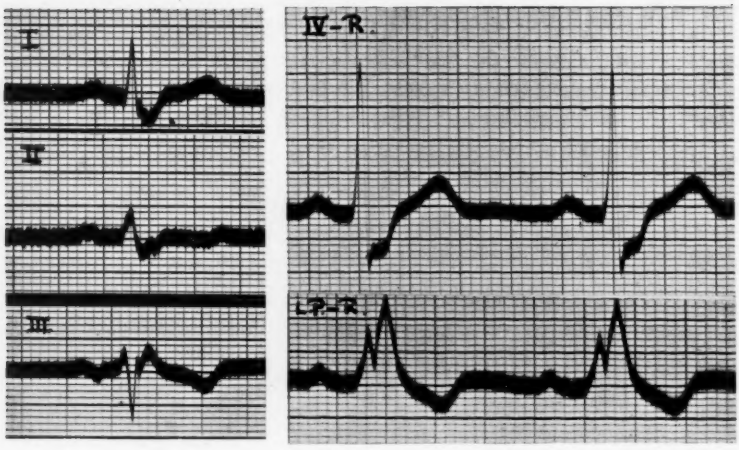
(A) Limb leads.

(B) Chest leads paired with the right arm.

Above : lead IV R showing a somewhat delayed intrinsic downward deflection and a diphasic T wave.

Below : the left pectoral lead (the right pectoral lead was similar) showing an early intrinsic downward deflection, a very deep S wave, and a very tall T wave.

It is, therefore, clear that for clinical work the pectoral leads are more valuable than lead IV for interpreting doubtful or bizarre forms of bundle branch block, and that the choice of distal electrode is a matter of indifference. It should be noted that when there is difficulty in distinguishing left bundle branch block from left axis deviation with inversion of T, no help can be obtained from chest leads ; but that when there is difficulty in distinguishing right bundle branch block from right axis deviation with inversion of T3, the left pectoral lead usually makes correct interpretation easy. Reference to the findings in cases of considerable left and right ventricular enlargement will clarify these remarks.



A B

FIG. 12.—Right bundle branch block—common type (new terminology).

(A) Limb leads.

(B) Chest leads paired with the right arm.

Above : lead IV R showing a normally timed intrinsic downward deflection and gross slurring of S. The appearances are similar to those in lead I.

Below : the left pectoral lead (the right pectoral lead was similar) showing a notched upright QRS complex with a late intrinsic downward deflection, and an inverted T wave.

DIGITALIS

The effect of digitalis upon the limb lead electrocardiogram is well known. There is a characteristic depression of the RS-T segment. Strauss and Katz (1935) using chest leads came to the conclusion that either elevation or depression of the RS-T segment might occur. They used the old polarity. Their illustrations of elevation of the RS-T segment were convincing, but the same cannot be said of their example of depression of that segment.

Stewart and Watson (1938) have also investigated the effect of digitalis upon the chest lead electrocardiogram. Using an anterior-posterior lead and the old polarity they found that elevation of the RS-T segment occurred in the majority of cases, but that occasionally there were other effects. The electrocardiograms showing these other effects, which included depression of the RS-T segment, were bizarre.

Holzmann (1937), using an anterior-posterior chest lead and the new polarity, found that digitalis commonly caused depression of the RS-T segment similar in character to that seen in limb lead electrocardiograms. Occasionally, however, especially when there was eccentric hypertrophy of the left ventricle or of both ventricles, digitalis produced a dome shaped RS-T segment which was elevated to a maximum of 0·2 mv. above the iso-potential level. These curves resembled those of myocardial infarction, and correct interpretation

was sometimes difficult. When elevation exceeded 0·2 mv., a digitalis effect could be excluded.

We have used chest leads in fifteen cases treated with digitalis. Depression of the RS-T segment, similar in character to that seen with limb leads, and often in an exaggerated form, was found in every instance. It was most pronounced in lead IV except in those cases with marked right ventricular dominance when it was most evident in the right pectoral lead (see Fig. 13). It was shown rather

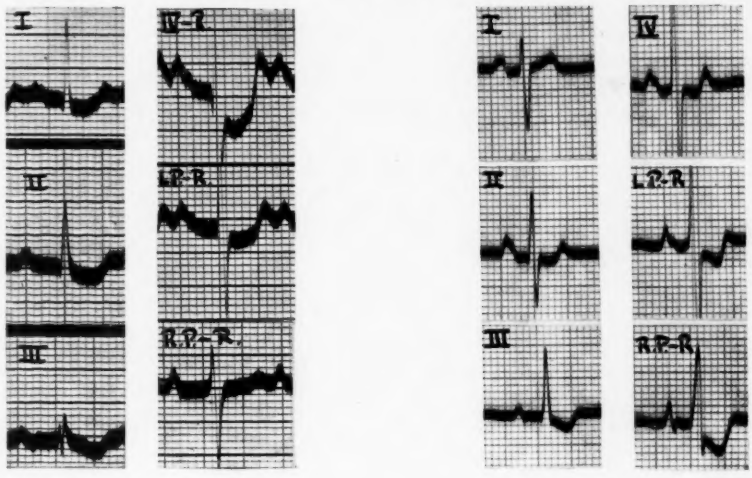


FIG. 13.—Limb leads (A), and chest leads paired with the right arm (B), in a case of digitalis intoxication. There is very marked depression of the RS-T segment in lead IV, but none in the right pectoral lead. Case of thyrotoxicosis.

(C), (D) Limb leads, and chest leads paired with the right arm, in a case of marked right ventricular dominance due to mitral stenosis, pulmonary hypertension and probable pulmonary embolism. The digitalis effect is most marked in the right pectoral lead.

better when the proximal electrode was paired with the right arm than when it was paired with the left leg.

It is concluded that lead IV R is suitable for showing the effect of digitalis except in cases with marked right ventricular dominance when the right pectoral lead should be used, and that when there is doubt as to the proper interpretation of a possible digitalis effect with limb leads, chest leads may be helpful by showing this effect in an exaggerated form.

MYOCARDIAL INFARCTION

Wood and Wolferth (1932, 1933) showed that an anterior-posterior chest lead electrocardiogram would produce typical changes in the RS-T segment after experimental ligature of the anterior descending branch of the left coronary artery of the dog, when the limb lead electrocardiogram was normal. They concluded that this might also apply to certain cases of myocardial infarction in

man. An opportunity to test this suggestion soon presented itself. A case, clinically diagnosed as myocardial infarction, failed to show changes with the limb leads. An anterior-posterior chest lead was employed, and the electrocardiogram so obtained showed a remarkable deviation of the RS-T segment from the iso-potential level. This case subsequently exhibited diagnostic changes in the limb leads. To it was added another case which showed typical RS-T changes in both limb and chest leads, and then with thirty-three controls the facts were published (Wolferth and Wood, 1932 a) prior to the experimental reports given above. Shortly afterwards they published a short article (1932 b) describing three cases of myocardial infarction in which the changes were more evident in limb than in chest leads. One of these cases exhibited a typical T1 type of coronary curve, but the other two were probably both posterior infarctions.

Several hasty reports by other authors followed and have been sharply criticized by Roth (1935). But in the last six years certain facts have become established. In anterior myocardial infarction changes occur in the QRS complex, in the RS-T segment and in the T wave, and any one of these changes may be shown only or best in one particular lead either limb or præcordial. Wilson and his associates have made a special study of the behaviour of the QRS complex with limb (Wilson and others, 1932 a, 1933) and with semi-direct leads (Johnston and others, 1935 ; Wilson and others, 1932 a, 1935 a, 1935 b, 1933), both in the dog and in man. They have shown experimentally that monophasic downward deflections (new polarity) are obtained from areas underlying which there is infarction of the whole thickness of the ventricular wall, but that at the edges of the infarct where there is living tissue, or over the centre of the infarct if it only involves part of the thickness of the ventricular wall, a W complex is usual. Semi-direct leads taken from points overlying healthy muscle away from the infarct yield normal QRS complexes. These QRS changes may be transient or permanent according to the degree of recovery of the muscle deprived of its blood supply. In clinical cases they found that QRS changes were more likely to be permanent than T wave changes.

The occurrence of a monophasic downward deflection in the chest lead in cases of anterior myocardial infarction has been well established (Bohning and Katz, 1938 ; Donzelot and Pelaez, 1938 ; Faulkner, 1936 ; Feinstein and Lieberson, 1937 ; Freeman, 1937 ; Goldbloom, 1934 ; Holzmann, 1937 ; Jervell, 1935 ; Master and other, 1937 ; Wood and others, 1933), but little attention has been paid to the W complex. Amongst the authors who stress the importance of a monophasic downward deflection are Master, Dack, Kalter and Jaffe (1937) who also state that a very small R wave is significant. They found it in 37 per cent. of cases with ischæmic heart disease compared with the figure of 7·5 per cent. for normal controls. When one considers that about 30 per cent. of cases with ischæmic heart disease have considerable enlargement of the left ventricle from hypertension or from syphilitic aortic incompetence, it is seen that the figure of 37 per cent. is exactly what one would predict, for we have already shown that cases with considerable enlargement of the left ventricle

ordinarily have a very small R wave in the left pectoral lead which is more or less the lead they used.

We have analysed the QRS complex in eighteen cases of anterior myocardial infarction. In lead IV R or F we found a W complex in no less than twelve cases, and a monophasic downward deflection in only three. The shape of the W varied considerably, but so long as there were three conspicuous deflections, Q, R and S, we included it. In the left and right pectoral leads QRS was represented by a monophasic downward deflection in fifteen cases. Eight patients were followed for periods of six months to two and a half years : the W complex was permanent in three and transient in two ; the monophasic downward deflection was permanent in five and transient in two.

The finding of W complexes in apical leads and monophasic downward deflections in the pectoral leads, which have also been recorded by Kossmann and de la Chapelle (1938), is a little difficult to understand. According to the experimental work of Wilson and his associates already mentioned, monophasic downward deflections might be expected in lead IV over the site of the infarct, and W complexes in the left pectoral lead at the edge of the infarct, in the common cases of anterior apical myocardial infarction. If the infarct should involve only the inner part of the muscle wall, the W complex would be understood at the apex, but in that case why should there be a monophasic downward deflection in the left pectoral lead ?

Elevation of the RS-T segment is commonly seen in the early stages of anterior myocardial infarction, and has frequently been described (Bohning and Katz, 1938 ; Donzelot and Pelaez, 1938 ; Feinstein and Lieberson, 1937 ; Freeman, 1937 ; Holzmann, 1937 ; Jervell, 1935 ; Lieberson and Liberson, 1933 ; Swan, 1937 ; Van Neiuwenhuizen and Hartog, 1937 a ; Willcox and Lovibond, 1937 ; Wilson, 1936) since the article by Wolferth and Wood (1932). In our series elevation of the RS-T segment occurred in nine out of twelve cases which were investigated sufficiently early. It was always evident in lead IV and in the left pectoral leads, but it only occurred in the right pectoral lead in four cases. The choice of distal electrode was a matter of indifference. As with limb leads this change was always transient.

Subsequent inversion of the T wave has been described by practically all authors on the subject of chest leads in myocardial infarction, and Wolferth and Wood (1933) have reported seven cases in which these T waves were " huge." All our cases showed subsequent inversion of the T wave in the apical leads, and all but three in the left pectoral leads. In the right pectoral—right arm lead the T wave was inverted in five cases, and in the right pectoral—left leg lead in seven. Of eight cases followed for a sufficient period of time the inversion of the T wave was permanent in four cases in the apical leads, in three cases in the left pectoral leads, and in four cases it was transient.

Characteristic curves showing the W complex, the monophasic downward deflection, the elevation of the RS-T segment, and the inverted T wave are shown in Fig. 14.

There have been different reports concerning the findings with chest leads in cases of posterior myocardial infarction. All are agreed that the QRS

complex is not affected, and that the T wave is not inverted. Though some (Jervell, 1935 ; Swan, 1937 ; Wood and others, 1933) have concluded that the RS-T segment is usually unaffected, others have shown that there may be marked depression of this segment in the early stage of posterior infarction (Bohning

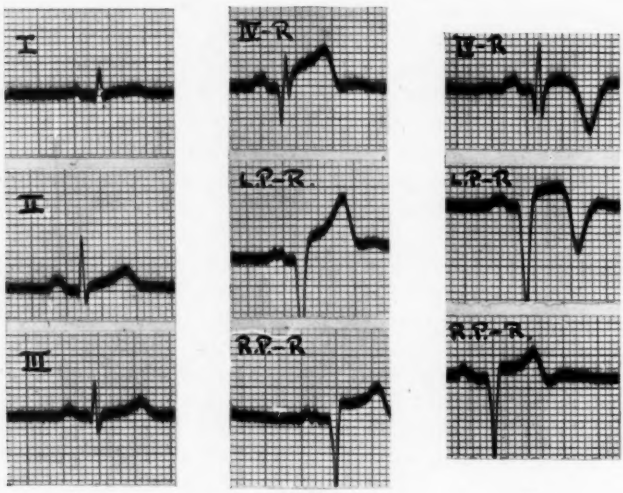


FIG. 14.—From a case of anterior myocardial infarction.

Limb leads (A), and chest leads paired with the right arm (B), showing a deformed W complex in lead IV, monophasic downward deflections in the pectoral leads, and elevation of the RS-T segment in all chest leads.

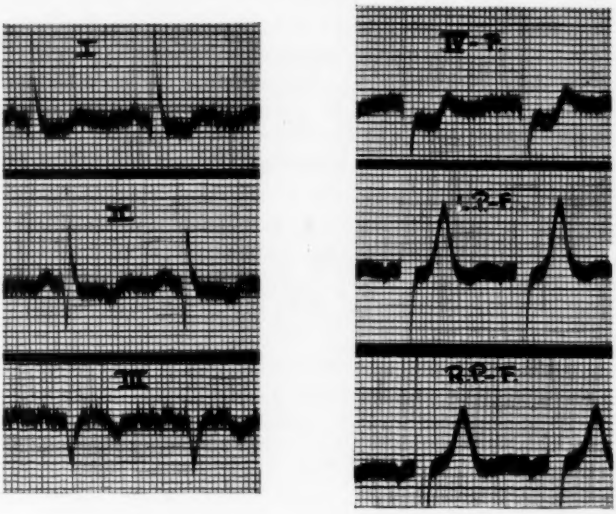
Chest leads paired with the right arm (C), a week later, showing a typical W complex in lead IV, monophasic downward deflections in the pectoral leads, somewhat less elevation of the RS-T segment in all chest leads, and sharp inversion of the T wave in lead IV and in the left pectoral lead.

and Katz, 1938 ; Feinstein and Lieberson, 1937 ; Holzmann, 1937 ; Willcox and Lovibond, 1937 ; Wilson and others, 1932 a). It has also been shown that as the RS-T segment approaches or resumes the iso-potential level, the T wave may become very tall (Bohning and Katz, 1938 ; Feinstein and Lieberson, 1937 ; Willcox and Lovibond, 1937 ; Wilson and others, 1932 a).

Fifteen cases of posterior myocardial infarction were included in our series. QRS was normal in all instances. The RS-T segment was depressed in the apical leads in eight out of ten cases which were investigated sufficiently early. This depression was not so evident in the left pectoral leads and did not occur at all in the right pectoral leads. Inversion of the T wave did not occur, but in three instances an upright T wave of very high voltage succeeded the early depression of the RS-T segment. The distal electrode did not materially influence any of these findings (see Fig. 15).

The characteristic electrocardiographic changes of anterior myocardial infarction are very rarely restricted to chest leads if serial records are obtained,

but they may occur earlier in chest leads, where they may be diagnostic when still of doubtful significance in limb leads, and they may last longer in chest leads. Very occasionally an infarct may affect lead I and not the chest leads.



A B

FIG. 15.—From a case of posterior myocardial infarction.

(A) Limb leads.
(B) Chest leads paired with the left leg, showing depression of the RS-T segment in lead IV, and tall T waves in the pectoral leads.

Wolferth and Wood (1932 b) reported such a case, and we have recently seen an example.

In our series of thirty-three cases of myocardial infarction serial limb leads were diagnostic of the condition in all but one instance, but in four others the changes occurred earlier in the chest lead graphs so that a correct diagnosis was made sooner. We derived no help from the chest leads in the cases of posterior myocardial infarction, for in these the limb leads required no support, and the chest leads had but little to give.

Before concluding, reference must be made to the value of chest leads in cases of multiple infarction. Wolferth and Wood (1935), and Feinstein and Lieberson (1937) have drawn attention to this.

A combination of anterior and posterior myocardial infarction may give rise to an atypical limb lead electrocardiogram which causes difficulty in diagnosis. In this event, chest leads may prove of use by unmasking the anterior infarct. The atypical electrocardiograms of the two cases which came to autopsy in the paper by Wolferth and Wood (1935) were examples of the T_2 pattern described by Wood (1937) in cases of pericarditis and hæmopericardium, and it is therefore unfortunate that no statement was made as to the

presence or absence of pericarditis in the two cases mentioned. However, there can be little doubt as to the truth of their contention, and we have recently seen a good example in which limb leads were atypical but suggested posterior myocardial infarction, whereas chest leads were diagnostic of anterior infarction. Autopsy showed a large infarct at the apex of the left ventricle anteriorly, but spreading round to involve a considerable portion of the posterior wall.

Summary of the Effects of Myocardial Infarction on the Chest Lead Electrocardiogram

A. In anterior myocardial infarction

1. QRS is often represented by a W complex in the apical leads, and by a monophasic downward deflection in the pectoral leads.
2. Initial elevation of the RS-T segment and subsequent inversion of the T wave commonly occur in both apical and left pectoral leads but are less evident in the right pectoral leads.
3. Any one of these changes, except elevation of the RS-T segment which is always transient, may or may not be permanent.
4. Both the right arm and the left leg are entirely satisfactory distal electrodes for the demonstration of these changes.

B. In posterior myocardial infarction

1. The QRS complex is normal.
2. Initial depression of the RS-T segment is common, and is most evident in the apical leads.
3. Very tall upright T waves are sometimes seen in the later stage either in the apical or in the left pectoral leads or in both.

For clinical work, chest leads are of undoubted value in cases of suspected myocardial infarction, especially by enabling one to make an earlier diagnosis in acute cases, and a correct diagnosis in cases seen for the first time some months after the event.

Angina Pectoris

Chest leads have been considered helpful in the diagnosis of angina pectoris. Thus H. D. and S. A. Levine (1936), using a left para-sternal—left leg lead in addition to lead IV F, found that 16 per cent. of their cases of angina pectoris lacked an R wave. In another series of forty-four autopsy cases they showed that an absent R wave in lead IV F was associated with myocardial infarction in twelve out of fifteen cases. Two of the other three had bundle branch block, and the third had tuberculous pericarditis. (To avoid confusion we have translated their nomenclature into the current terminology.) They concluded that an absent R wave in the leads they used was important evidence of ischæmic heart disease.

Willcox and Lovibond (1937), preferred a left para-sternal—right arm lead. They investigated sixty-two cases of angina pectoris. In thirty-four the

electrocardiogram was normal ; in eleven both limb and chest leads were abnormal ; in nine the limb leads were abnormal, but the chest lead was normal ; and in eight the limb leads were normal, but the chest lead abnormal. The abnormalities in the chest leads consisted of very small or absent R waves and inversion of the T waves.

Edeiken, Wolferth and Wood (1936) considered that reversal of the T wave in their antero-posterior chest lead should be considered pathological when it was the only abnormality of the adult electrocardiogram. In such cases they suspected coronary artery disease.

The chest lead electrocardiograms in cases of angina pectoris must be interpreted with caution. For if there is associated hypertensive heart disease or aortic valvular disease with much enlargement of the left ventricle, an inverted T wave in the apical leads and a very small R wave in the pectoral leads are only to be expected, and cannot be used to support the diagnosis of angina pectoris. There were twenty-seven cases of angina pectoris in our series. Only three of these showed chest lead abnormalities which could not be accounted for by the known underlying condition, and in these three there was inversion of the T wave in leads IV R and F but not in the pectoral leads. The limb leads of these three cases were normal.

It is concluded that chest leads may be of service in giving support to the diagnosis of angina pectoris by revealing the presence of previous myocardial infarction. Both lead IV and the left pectoral leads are therefore advised, the former to show W complexes, the latter monophasic downward deflections, and either or both, inverted T waves.

DISCUSSION

The Distal Electrode

The choice of distal electrode lies between the dorsal electrode originally used by Wolferth and Wood (1932 a), the neutral electrode devised by Wilson and his associates (1934 d), and one of the limb electrodes. Because the dorsal electrode is inconvenient to apply, and because it has not been shown to have any advantage over the others, it seems wise to discard it. We have had no experience of Wilson's neutral electrode, but because it requires additional apparatus it is unlikely to meet with much favour in clinical work unless it proves to have definite advantages, and so far there is little evidence of this. Keller (1937), after comparing the neutral electrode with the right arm electrode used by Groedel (1934), came to the conclusion that the latter was preferable both on theoretical and practical grounds. The majority of workers now use one of the limb electrodes, and the most favoured are the left leg and the right arm. The left leg has been more widely used for two reasons : first, because the technique was simpler with the old polarity, and secondly because more work had been done with this electrode on normal controls. Neither of these reasons is now valid. With the new polarity the technique is simpler with the right arm electrode, and adequate work has now been done with this electrode

on normal controls. The choice between these two electrodes should be made on their respective merits.

If only one chest lead is used, lead IV (the apical lead) has been recommended by the Cardiac Society of Great Britain and Ireland and by the American Heart Association. Now in lead IV, there is only one difference between the distal electrodes under consideration : in young children lead IV F may occasionally show a diphasic T wave (Sorsky and Wood, 1937), and if the proximal electrode is placed slightly inside the apex beat the T wave may be inverted, whereas lead IV R exhibits an upright T wave in normal controls of any age, and the proximal electrode may be placed well inside the apex beat without altering the direction of the T wave. Since lead IV R is the simpler lead with the new polarity, it has two advantages and no disadvantages, and we, therefore, recommend it without hesitation.

If multiple chest leads are used the problem becomes more complicated. The left leg has the advantage when chest leads are used to distinguish left ventricular enlargement from cardiac displacement due to a high diaphragm, for we have shown that in the right pectoral—left leg lead an inverted T wave is incompatible with considerable left ventricular enlargement, but is common in normal controls and in cases of cardiac displacement due to a high diaphragm. The right arm distal electrode is useless for this purpose. We have found no other major advantage in the left leg distal electrode, but there is one other minor advantage : if R is taller than S in the right pectoral—left leg lead, it may be concluded that there is dominance of the right ventricle. A similar finding in the right pectoral—right arm lead is suggestive but less certain.

The right arm distal electrode has been used extensively by Groedel (1934), was independently suggested by Roth (1935), advised by Larsen (1937), and favoured by Willcox and Lovibond (1937). Apart from its convenience it has two considerable advantages : first, normal standards are more uniform in that the T wave remains upright in all leads in adults, and is only rarely inverted in children and then only in the right pectoral lead ; as a corollary, inversion of the T wave in the pectoral leads has much more significance with the right arm distal electrode than it has with the left leg ; secondly, the P wave is upright and of adequate dimensions in leads paired with the right arm, whereas it is usually inverted and is very small in leads paired with the left leg. Although we have not drawn attention to the value of the right pectoral lead in studying disorders of rhythm, it may be remembered that Lewis (1909–10) found it expedient to use such a lead in his study of auricular fibrillation. Holzmann (1937) found that abnormalities of the P wave could be well shown by placing the exploring electrode over the right auricle, and we have been using the right pectoral—right arm lead to demonstrate the tall sharp P wave associated with right auricular enlargement. There can be little doubt that the right arm is the better distal electrode for these purposes.

We conclude that the right arm is the distal electrode of choice for single or multiple chest leads for routine purposes, but for the special purpose of distinguishing left ventricular enlargement from cardiac displacement due to a high diaphragm we recommend the right pectoral—left leg lead.

Multiple Chest Leads

The Cardiac Society of Great Britain and Ireland recommended a single chest lead, lead IV, for routine use, because it was considered that there was insufficient evidence upon which to recommend multiple chest leads.

Wilson and his associates, who have employed the multiple chest leads recommended by the American Heart Association, have concluded that anterior myocardial infarction will rarely escape detection if multiple chest leads are used in addition to the limb leads.

Holzmann (1937) used the chest leads in all types of heart disease, and found them useful in conditions other than myocardial infarction and angina pectoris. He advised the use of both the apical and the right pectoral lead, the latter being of aid in the diagnosis of right ventricular enlargement. Hecht (1936) came to a similar conclusion.

Groedel (1934) claims to have isolated the lævocardiogram and the dextrocardiogram. The former is obtained with lead IV R, and the latter with a left sternal—right arm lead. Whether his theoretical considerations are right or wrong it is clear that considerable information may be gained by employing both these leads.

There can be no doubt that lead IV is an essential chest lead because it may be the only one to show evidence of myocardial infarction, especially by exhibiting a W complex or an inverted T wave. It is also certain that a monophasic downward initial ventricular complex indicating myocardial infarction may be restricted to the pectoral leads, and that, therefore, the left pectoral or *a* left pectoral lead is essential because this finding loses some of its significance in the right pectoral lead. In acute anterior myocardial infarction elevation of the RS-T segment is usually seen in both apical and left pectoral leads, but may be restricted to one or the other. If chest leads were only used in cases of possible ischæmic heart disease it is clear that both lead IV and a left pectoral lead should be employed.

But chest leads may be of value in other conditions such as rheumatic carditis, pericarditis, acute cor pulmonale from pulmonary embolism, left and right ventricular enlargement, bundle branch block, and for studying the form of the P wave and certain abnormalities of rhythm. For these purposes the right pectoral lead, or a combination of the right pectoral lead and one or both of the others, is required.

It is concluded that at least three chest leads may be used with advantage, not only because each has a function not shared by the others, but also because additional information may be gained by studying them in relation to one another.

The three chest leads which we have used were suggested by Roth (1935), and we have found them satisfactory. There are, however, two other favoured combinations of leads : the first is that used by Groedel (1934) and by Hecht (1936), and consists of two leads, one apical, the other basal, either just to the left of, or just to the right of the sternum, in the fourth intercostal space ; the second is that recommended by the American Heart Association and consists

of a series of leads from the following points—the right border of the sternum in the fourth intercostal space, the left border of the sternum at the same level, midway between the latter and the point next to be described, the mid-clavicular line in the fifth intercostal space, the anterior axillary line at the same horizontal level, and the mid-axillary line, these leads being numbered from 1 to 6 with their appropriate prescript according to the distal electrode employed. The prescripts used to denote the distal electrode employed are : for the right arm—CR, for the left leg—CF, for the neutral electrode devised by Wilson and his associates—V. Thus the right pectoral lead which we have used would be called lead CR 1 when it is paired with the right arm, and CF 1 when it is paired with the left leg ; our left pectoral leads would be called leads CR or CF 3 approximately ; and our apical leads would be usually leads CR or CF 4 or 5. The letter C stands for " chest."

The choice between these three different combinations of multiple chest leads is no easy one. We have sufficient evidence to justify the use of three rather than two chest leads as a routine procedure when chest leads are requested. Because we have only used three chest leads we are unable to express an opinion as to the value of more. When the apex beat is within the anterior axillary line it is probable that three leads would be sufficient ; when it is further to the left there may be some advantage in four or even five leads. When only three leads are used it would seem more rational to employ the three suggested by Roth (1935), as described in this paper, rather than to lead from three fixed points on the chest wall.

Summary and Conclusions

1. The normal appearances of electrocardiograms obtained with lead IV R have been presented.

2. Five other chest leads were investigated : lead IV F ; the left pectoral—right arm lead ; the left pectoral—left leg lead ; the right pectoral—right arm lead ; and the right pectoral—left leg lead.

3. Limb lead and multiple chest lead electrocardiograms were obtained from 302 selected cases of heart disease, and from twelve cases of cardiac displacement due to a high diaphragm.

4. In addition to their value as an aid in the recognition of ischæmic heart disease, chest leads have been found of service in the diagnosis of rheumatic carditis, pericarditis, left ventricular enlargement, right ventricular enlargement, acute cor pulmonale resulting from pulmonary embolism, and of doubtful or bizarre forms of bundle branch block.

5. The right arm was both the most informative and the most convenient site for the distal electrode ; and we believe that the evidence is now sufficient to warrant its routine use in preference to the left leg. The latter was only advantageous when it was desired to distinguish left ventricular enlargement from cardiac displacement due to elevation of the diaphragm.

6. Multiple chest leads are of value not only because they may yield

diagnostic evidence of myocardial infarction when a single chest lead is barren, but also because they are a safeguard against faulty interpretation of lead IV, because each of the three leads (lead IV and the two pectoral leads) used in this investigation has some special value peculiar to itself, and because a study of these three leads in relation to one another may give information which is unobtainable by a study of individual leads.

Our thanks are due to Sir Frederick Menzies, chief medical officer of the London County Council, for his permission to publish this material, and to Prof. F. R. Fraser for his constant encouragement and advice.

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A NEW SIGN OF LEFT VENTRICULAR FAILURE

BY

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The prominent P wave associated with mitral stenosis is well recognized ; it is usually widened and is often notched, bifid, or flat-topped ; it is generally thought to be a result of left auricular hypertrophy. There is also some evidence that a tall spiked P wave may be produced by right auricular hypertrophy, for such a wave is rarely found except in cases of tricuspid stenosis, congenital pulmonary stenosis, and chronic cor pulmonale. It is the purpose of this communication to draw attention to a third type of P wave which we have found to be commonly associated with hypertensive heart disease, especially when there is left ventricular failure. This P wave is widened and of low voltage ; it is also usually bifid or flat-topped. Typical examples of these three types of P wave are shown in Figs. 1–3.

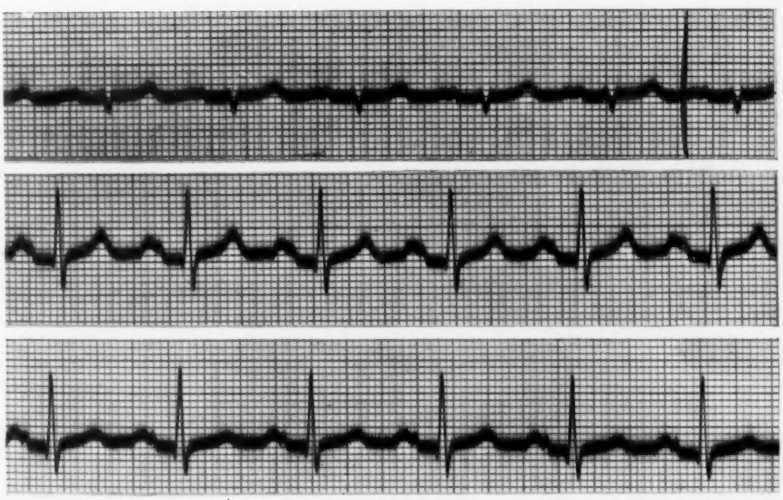

FIG. 1.—Prominent bifid and widened P wave in a case of mitral stenosis.

† Received July 28, 1938.

The normal P wave may be notched, but its duration does not exceed 0·1 sec. Its voltage in the favourable lead rarely exceeds 0·25 mv. and averages a

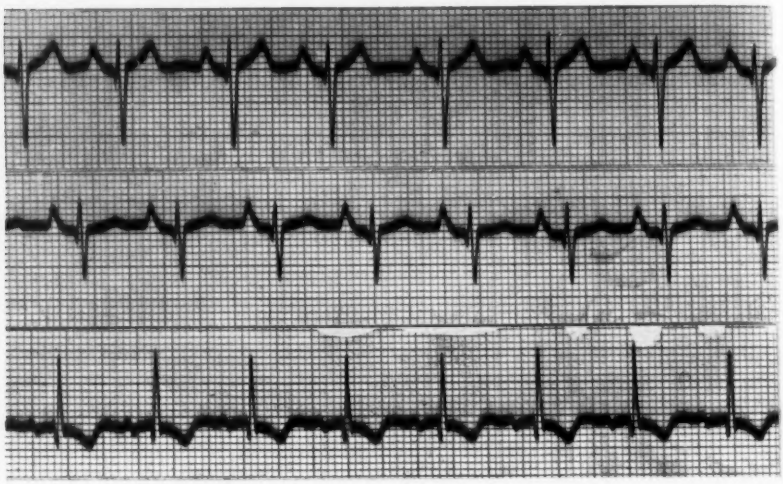

FIG. 2.—Tall spiked P wave in a case of chronic cor pulmonale (proved at autopsy).

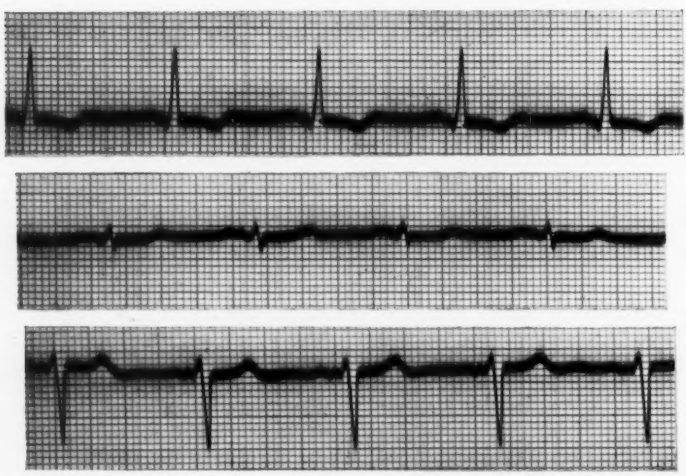

FIG. 3.—Wide bifid P wave of the low voltage in a case of hypertensive heart disease with left ventricular failure.

little under 0·15 mv. The P wave to which we are drawing attention commonly measures 0·12 sec. in duration and rarely more than 0·1 mv. in amplitude.

In a series of 1000 consecutive routine electrocardiograms this P wave was

found in 25 instances, or in 2·5 per cent. Four of these were cases of aortic valvular disease, and the remainder were cases of hypertensive heart disease. In the same series there were 25 instances of abnormal P waves associated with mitral stenosis, and five instances of tall spiked P waves with chronic cor pulmonale or with congenital pulmonary stenosis. It is therefore evident that this P wave is as common as the P wave of mitral stenosis, and about five times as common as the tall spiked P wave associated with chronic cor pulmonale or with congenital pulmonary stenosis.

To determine the significance of this P wave, we studied 70 patients with hypertensive heart disease and 25 with uncomplicated aortic valvular disease in various stages of development. As a control group we used 25 patients with ischæmic heart disease without cardiac enlargement. They were examined clinically, radiologically, and electrocardiographically, and no one was included where the diagnosis was in doubt. We divided the patients with hypertensive heart disease and those with aortic valvular disease into five groups which were determined as follows :

Group A .. Those with no symptoms.
Group B .. Those with slight effort dyspnœa.
Group C .. Those with moderate or severe effort dyspnœa.
Group D .. Those with left ventricular failure.
Group E .. Those with right ventricular failure.

The diagnosis of left ventricular failure was made upon the presence of several of the following criteria : orthopnœa, paroxysmal cardiac dyspnœa, pulmonary œdema, persistent bilateral basal pulmonary rales, prolongation of the arm to tongue circulation time, radiological evidence of pulmonary congestion, pulsus alternans and gallop rhythm without bundle branch block. The diagnosis of right ventricular failure was made upon the finding of engorgement of the veins of the neck with enlargement and tenderness of the liver. The presence of dropsy was not considered essential for the diagnosis of right ventricular failure, nor was the latter diagnosis necessarily made because there was dropsy. Of course, all patients with failure of the right ventricle also had failure of the left ventricle.

The electrocardiograms were then analysed with regard to the P waves. These were classified as normal if they lasted for less than 0·1 sec., as doubtful if from 0·1 to 0·11 sec., and as widened if they exceeded 0·11 sec. We considered that the width of the P wave was the essential feature. Widened P waves occurred most frequently in lead II, but were often found in more than one lead. In some cases the bifid nature of P was so marked as to warrant the term " double P wave," for it consisted of two separate deflections, the one immediately following the other and each measuring 0·06 sec. in duration.

The results of this study are presented in Table I. In the hypertensive cases the incidence of a widened P wave increased from zero in the symptomless group to 61 per cent. in the group with left and right ventricular failure. It is important to note that there was no real difference in the incidence of a widened P wave in the group with left ventricular failure alone and the group with left and

right ventricular failure. Similar, but less convincing, results were obtained in the cases of aortic valvular disease. In the control group of 25 cases of ischæmic heart disease a widened P wave did not occur.

TABLE I.—RELATIONSHIP OF WIDTH OF P AND DEGREE OF CARDIAC FAILURE

	CLASS	P WAVE			NO. OF CASES
		>0·11″	0·1—0·11″	<0·1″	
Hypertensive heart disease ..	A	—	—	6 (100%)	6
	B	1 (4%)	—	23 (96%)	24
	C	2 (15%)	4 (30%)	7 (55%)	13
	D	7 (50%)	2 (14%)	5 (36%)	14
	E	8 (61%)	1 (8%)	4 (31%)	13
Aortic valvular disease ..	A	—	—	—	0
	B	—	2	1	3
	C	—	2	5	7
	D	3	2	3	8
	E	1	—	4	5
Ischæmic heart disease		—	2	23	25

It is evident that the type of P wave which we have described is commonly associated with left ventricular failure.

DISCUSSION

The cause of the abnormal P waves in mitral stenosis has been discussed by several authors. Although von Boros and Trendelenburg believe that an intra-auricular conduction defect is responsible, the majority think that the explanation lies in hypertrophy of the left auricle. Some attempts have been made to diagnose from the lead in which the notched P wave occurs (Winternitz) or from the shape of the notching (Holzmann) which auricle is affected, but there is insufficient evidence for these views. In a recent study, Berliner and Master tried to correlate electrocardiographic and post-mortem findings in cases of mitral stenosis. They found hypertrophy of the left auricle in cases with bifid and widened P waves, and hypertrophy of the right auricle in addition in cases in which the P wave was increased in height. Their tallest P waves occurred in cases of combined mitral and tricuspid stenosis. They suggested that a tall P wave might result from a summation effect from hypertrophy of both auricles. Because we are familiar with the tall spiked P wave associated with pulmonary stenosis and chronic cor pulmonale, and because we have noticed that, though tall, the P wave is not widened in these conditions, we suggest that an increase in voltage of the P wave may indicate hypertrophy of the right auricle, whereas widening and notching of the P wave may indicate hypertrophy of the left auricle.

If this view should prove correct, then there is a ready explanation for the wide bifid P wave associated with left ventricular failure, for in this condition an

increased stress falls upon the left auricle. It is tempting to suggest that this type of P wave is yet another sign, and an early one, of left ventricular failure, to be ranked with gallop rhythm.

This is but a preliminary report and is published in the hope that it may stimulate investigation in this field. With the aid of an œsophageal lead, a semi-direct right auricular lead, and careful clinical and post-mortem investigation it should not be difficult to solve the problem. We already have evidence that chest leads taken from the right border of the sternum and paired with the right arm electrode may yield a tall spiked narrow P wave in cases with hypertrophy of the right auricle.

Summary

1. A widened P wave of low voltage, usually bifid or flat-topped, has been found in association with left ventricular failure in cases of hypertensive heart disease and of aortic incompetence.

2. It is suggested that this P wave results from left auricular stress.

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THE SIGNIFICANCE OF ELECTROCARDIOGRAMS SHOWING A "SECOND POSITIVE WAVE OF QRS" IN LEAD III

BY

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During the past two years an investigation has been made to determine whether electrocardiograms showing anomalous forms of QRS in lead III have any diagnostic or prognostic importance. Several distinct types of anomalous QRS3 have been encountered, and the findings in one of these types have already been published ("Large Q3," Peel, 1938). It is now proposed to consider a second variety in which the R and S deflections are followed by a third wave directed upwards and pointed. Katz and Slater (1935) have named this "the second positive wave of QRS3" and specify the following criteria for its recognition :

1. The standardization must show no "overshoot."
2. R3 must be a positive wave above the iso-electric line.
3. R3 must be followed by a definite S3, the amplitude of which may vary.
4. The upstroke of S3 must rise above the iso-electric level to a positive peak, and then descend either suddenly or gradually to the iso-electric level ; this peak may or may not be notched.

Katz and Slater collected 320 examples from a series of 8000 electrocardiograms and published a preliminary report on 50 of them, including post-mortem records in three cases. Clinical evidence of organic disease was present in 86 per cent., and they concluded that the abnormality should be regarded as indicative of organic myocardial disease.

Hope Gosse and Lowe (1937) found complexes of this type in 2·2 per cent. of all their cases, and published an analysis of 44 ; they arrived at a conclusion diametrically opposed to that of Katz and Slater : "Of this 2·2 per cent. the cases were almost equally divided between hearts diagnosed as clinically normal and those in which myocardial damage was considered to be present. . . . As a result of this investigation we do not find that any diagnostic importance can as yet be placed upon this variation from the average normal ventricular complex."

Although these are the only publications which deal directly with the type

† Received June 2, 1938.

of complex in question, certain others have an indirect bearing on the subject. Katz and Slater admit any electrocardiogram in which lead III fulfils their requirements, irrespective of the presence or absence of abnormality in the remaining leads and irrespective of the breadth of the QRS complex. Some electrocardiograms which have been described as examples of right branch bundle block conform to their definition in so far as QRS3 is concerned, though it is true that Wilson's (1934) original description of what has since been termed " the common variety of right branch bundle block " fails to fulfil their second condition, as there is no initial R3. V. Deesten and Dolganos (1934), however, include some records with and some without an initial R in their description of " atypical branch bundle block with a favourable prognosis " ; those with an initial R satisfy all of Katz and Slater's criteria. It is of interest to note that the single illustration published by Hope Gosse and Lowe has a broad QRS measuring 0·12 sec., with a broad S in leads I and II corresponding in time to the " second positive wave " of lead III ; it differs from Wilson's description of branch bundle block only in the presence of the initial R3.

Another article germane to the subject is by Bland and White (1931), entitled " The clinical significance of complete inversion of lead III." Admittedly the electrocardiogram illustrated shows no " second positive wave " ; it has inverted P and T waves, small R and large S. It is clear, however, that the appearance of the QRS complex resulting from inversion will vary from case to case according to the deflections originally present. A complex consisting of Q and R will become R and S after inversion ; one in which the original deflections are R and S will show Q and R ; while inversion of an electrocardiogram with Q, R, and S will produce a complex consisting of R, S, and a " second positive wave."

Bland and White had 115 cases which they considered to show complete inversion of lead III. Organic disease was only present in 55 per cent. as compared with 82 per cent. of their total patients. On the other hand, 72 per cent. of those with inversion of lead III had some condition liable to produce a transverse lie of the heart, and the abnormality was often abolished by deep inspiration. They conclude that an electrocardiogram showing complete inversion of lead III is usually associated with a transverse lie of the heart and has little clinical significance. Proger (1931) described electrocardiograms similar to those discussed by Bland and White as occurring frequently in obesity. It may be recalled that Hurxthal (1933) suggested that a " large Q3 " in many instances represented an " inverted R." We have previously shown (Peel, 1938) that electrocardiograms with a large Q3 can be subdivided into two groups, one of which is closely associated with coronary disease (Q2 present), while the other is frequently found in obese patients and is in many cases related to a transverse lie of the heart (Q2 absent). If Hurxthal's suggestion is accepted, the findings relative to this second group are brought into line with those of Bland and White.

It seems reasonable to suggest that the discrepancy between the conclusions of Katz and Slater and those of Hope Gosse and Lowe may to some extent be explained on the assumption that electrocardiograms showing a " second positive wave " in lead III are not all identical. Some may represent branch

bundle block, implying organic myocardial disease ; others may correspond to Bland and White's " inversion of lead III," and be associated with a transverse lie of the heart ; and there may well exist yet other types. Another possible explanation of the difference found in the two series may lie in the nature of the material from which the cases were drawn.

In dealing with the present series, we propose to use the symbol " R′ " for the " second positive wave of QRS ", examples of which are shown in Figs. 1 and 2. We have analysed 1200 consecutive cases from our original material

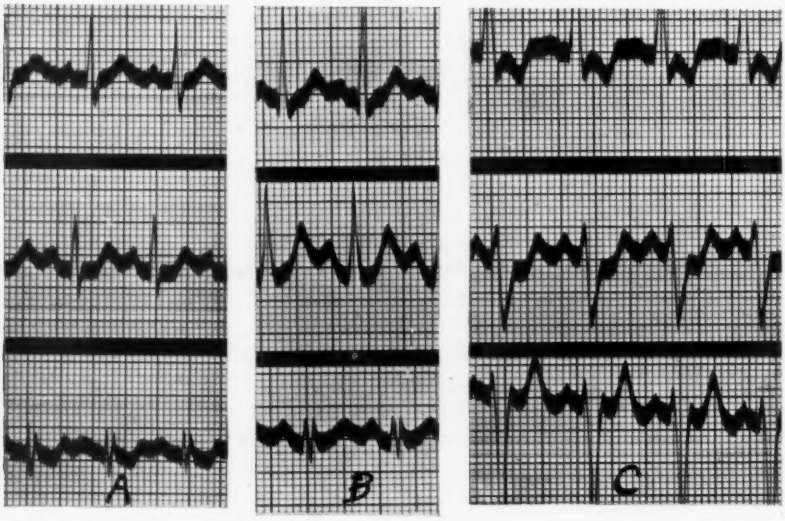

FIG. 1.—Examples of QRS3 showing a " second positive wave " and R3 exceeding 2 mm. in height. Type associated with a high incidence of organic disease and high mortality, irrespective of the presence of other electrocardiographic abnormality.

A. Electrocardiogram otherwise normal. Male, 41, hypertensive heart disease.
B. Electrocardiogram otherwise normal. Female, 21, mitral endocarditis.
C. Electrocardiogram otherwise abnormal. Male, 59, cardio-aortic syphilis and effort angina.

so as to compare the incidence of organic disease in patients with an " R′ " in lead III, with that in the total series. Next we have extracted those patients whose electrocardiograms were completely normal and compared them with those whose only electrocardiographic abnormality consisted of an " R′ " in lead III. We have investigated the influence of other features in the electrocardiogram in determining the diagnostic and prognostic significance of the abnormality. We have looked into the possibility of differentiating complexes with an " R′ " into two or more groups which might differ in their import. Finally in those of our cases in whom serial electrocardiograms are available, we have studied the conditions under which " R′ " has appeared or disappeared.

Analysis of Results

The material from which our examples were obtained consists of 1307 patients examined during the years 1928–1933. An analysis of 1200 consecu-

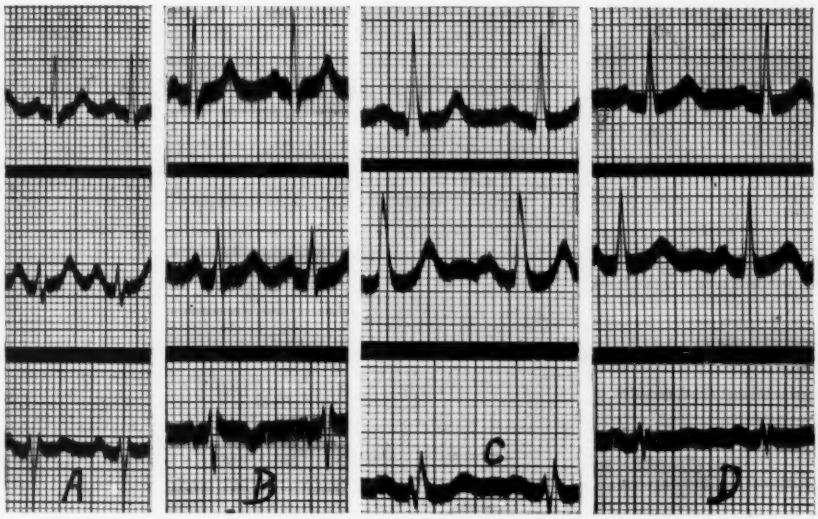

Fig. 2.—Examples of QRS3 showing a "second positive wave" and R3 small (less than 2 mm.) Type associated with a lower incidence of organic disease and low mortality provided the electrocardiogram is otherwise normal.

A. QRS3 resembling a normal complex which has been inverted, but P3 and T3 are upright. Male, 64, no organic heart disease.

B. QRS3 resembling an inverted normal complex ; P3 and T3 also inverted—"Complete inversion of lead III" (Bland and White, 1931). Female, 25, obese and neurotic.

C. QRS3 showing small R and S with large "second positive wave." A sparsely built man of 61 with a prominent abdomen.

D. QRS3 showing all three deflections small. Male, 61, effort syndrome, no organic heart disease.

tive cases showed 28 per cent. in whom no certain evidence of organic cardiovascular disease was detected clinically and 72 per cent. with such evidence. Of these, 33 per cent. had infective lesions ; 26 per cent. suffered from coronary disease, arteriosclerosis, or hypertension ; 4 per cent. had primary pulmonary disease, and 4 per cent. thyrotoxicosis ; the remaining 5 per cent. had miscellaneous conditions including myxœdema, obesity, anæmia with cardiac complications, congenital lesions, etc.

We have 62 patients in whom the electrocardiogram has at some time shown an "R′" in lead III, excluding one in whom it was only noted during paroxysms of ventricular tachycardia. Organic heart disease was recognized clinically in 51 cases (82 per cent.), a figure which agrees closely with that given by Katz and Slater. The incidence of organic disease is somewhat higher than in the total series, and the increase is confined to cases of coronary disease, arteriosclerosis, and hypertension ; these account for 57 per cent., or more than double their

incidence in the total material. Rheumatic lesions were less frequent (14·5 per cent. as compared with 28 per cent.), while pulmonary disease and thyrotoxicosis showed no difference.

Two patients were labelled " doubtful " and nine (14·5 per cent.) were thought to be free from organic cardiac disease at the time of examination. It is difficult to exclude heart disease with certainty in these cases. Five were below the age of 45 ; two of them had mild hyperthyroidism, one had extrasystoles, one had fainting attacks which were regarded as petit mal, and one was an obese woman with acute cholecystitis and a rapid pulse. The remaining six were over 45 ; two had extrasystoles, in one case following an acute infection and accompanied by tachycardia and impaired exercise tolerance ; on the other hand, two had a condition liable to produce a transverse lie of the heart (splenomegaly in one, diverticulosis in the other) ; and two suffered from a neurosis. It is significant that of the seven patients traced, three are working, a fourth is much improved, and the incapacity in one case is of nervous origin ; one died six years after examination from a cause unknown ; and the last died from acute circulatory failure following cholecystectomy. These cases may be summarized by stating that organic myocardial disease was probably present but unrecognized in one, that it may have been present in another who died, and that in three with extrasystoles there was a septic focus or recent infection which may have caused temporary myocardial damage. In the remaining six myocardial disease was probably absent ; two of them had a condition liable to produce a transverse lie of the heart.

Of the 62 patients with an " R′ " in lead III, 45 have been traced (March 1938). Twenty-five patients have died and three are incapacitated, but in one of them the incapacity is nervous in origin. Nine have improved and eight are working.

The Influence of other Electrocardiographic Features

Many electrocardiograms with an " R′ " in lead III show some other abnormality in addition (see Fig. 3), and these fall into two groups. The first includes findings which would be regarded as significant irrespective of the presence of " R′ " (e.g. RT displacement, coronary T wave, branch bundle block or intraventricular block, auricular flutter, and auricular fibrillation) ; the second includes those which would be considered of little or no importance in the absence of " R′ " (e.g. abnormalities of the electric axis, slight notching of auricular or ventricular complexes, digitalis effects, inversion of P3 or T3, extrasystoles, and nodal escape).

There are 26 cases in which the electrocardiogram showed some " significant " abnormality as defined above in addition to an " R′ " in lead III. Organic disease was present in all, and the prognosis was extremely bad. Out of 22 patients traced, 20 are dead, one is incapacitated, and only one has improved. In the two living patients, the " R′ " and the other abnormality were not present simultaneously, but succeeded one another. When an " R′ " and another significant abnormality were present simultaneously, all the cases traced have died (19 out of 23).

In 36 of our patients the electrocardiogram was normal except for the presence of an " R′ " in lead III. For comparison with these we have collected 352 cases from the same original material in whom the electrocardiogram was

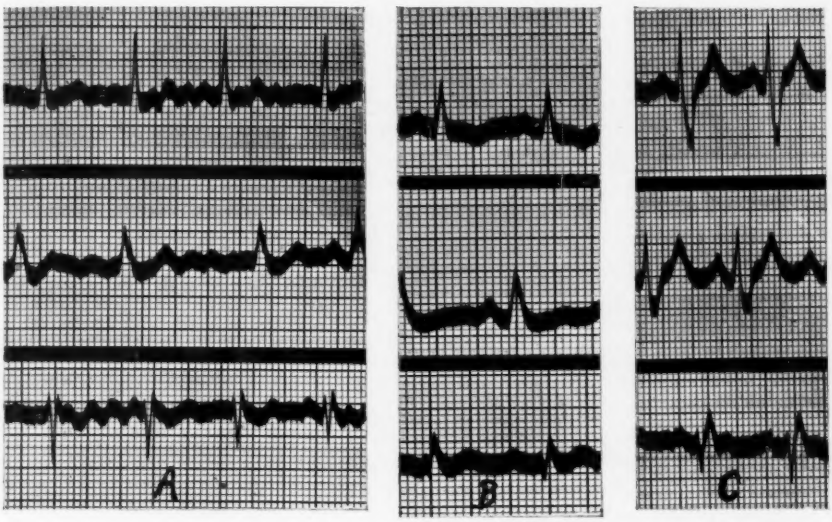

FIG. 3.—Examples of QRS3 showing a " second positive wave " and R3 small, but electrocardiogram otherwise abnormal. Type associated with high incidence of organic disease and high mortality.

A. QRS3 resembling an inverted normal complex. A sparsely built man of 59 with auricular fibrillation.

B. QRS3 with minute R and S and prominent " R′ " T1 inversion. Male, 62, hypertension.

C. Wilson's type of right bundle branch block, showing in lead III a small initial R, S, and broad " R′." The electrocardiogram fulfils Katz and Slater's criteria. Male, 57, with chronic coronary disease and congestive failure.

normal and showed no " R′ " in lead III. Organic heart disease is very much more frequent in those with an " R′3 " than in those without (70 per cent. as compared with 43 per cent.), and the increase is accounted for entirely by cases of coronary disease, of arteriosclerosis, or of hypertension ; these comprised 42 per cent. as compared with 8 per cent. in patients with normal electrocardiograms. Rheumatic lesions were slightly less frequent, while the incidence of other forms of heart disease was approximately the same.

We have no follow-up figures available for the patients whose electrocardiograms were completely normal. Of the 36 whose only abnormality was an " R′ " in lead III, 23 have been traced. Five are dead and two are incapacitated ; eight have improved and eight are able for work. The prognosis is very much better than when the electrocardiogram shows some " significant " abnormality in addition to an " R′ " in lead III.

We have attempted to determine whether there are any electrocardiographic features which will enable the diagnostic or prognostic value of an " R′ " to be

gauged when no other " significant " abnormality is present. If a relationship exists between some of these cases and Wilson's type of bundle branch block, it might be thought that the presence or absence of an S in lead I would help in their differentiation. This is not the case, there being no appreciable difference between the cases with and those without an S1. Having previously shown the importance of Q2 in assessing the diagnostic value of electrocardiograms with a " large Q3," it was thought that Q2 might also help in evaluating those with an " R′ " in lead III. There is, however, no significant difference in the incidence of organic lesions, the incidence of coronary disease, the mortality rate, or the recovery rate, when groups with and without a Q2 are compared.

There is evidence that the character of QRS3 itself may be of diagnostic and prognostic importance. Katz and Slater's definition is compatible with wide variations in the actual form of the complex; any one deflection (R, S, or " R′ ") may be prominent with the other two small ; any two may be prominent with the third small ; all three may be large, or all three may be small. Thus there are eight possible subgroups, each of which we have investigated individually. It was found that in every group with a prominent initial R (exceeding 2 mm. in height) organic disease was always present and the mortality was high—75 per cent. for the whole group and 50 per cent. even when " R′," was the only significant abnormality in the electrocardiogram (see Fig. 1). On the other hand in every group with a small initial R (less than 2 mm. in height) (see Fig. 2) there were a number of patients who were thought to be free from organic disease, and the mortality rate was much lower—45 per cent. for the whole group and only 12 per cent. when the electrocardiogram was otherwise normal. All eight patients who recovered working capacity and seven of the remaining nine who improved belong to the group with a small initial R and no other abnormality in the electrocardiogram.

This finding is of considerable importance in view of Bland and White's conclusions regarding " total inversion of lead III " ; for in one of the groups defined above, namely, that with a small R, large S, and small " R′," the complex exactly resembles a normal QRS which has been inverted ; P3 and T3 were only inverted in about half the cases. If it is justifiable to regard these as " inverted QRS complexes," the significance of the large initial R in the other group can be explained on the assumption that it represents inversion of a complex which originally showed a large Q3, a suggestion made by Hurxthal in 1933.

The electric axis is not strictly comparable with the height of R in lead III for some cases with a " prominent " initial R3 show left axial deviation, while some with a small R3 have a normal axis. When an " R′ " in lead III is the only abnormality in the electrocardiogram, there is little difference between those with a normal axis and those with left axial deviation, either in the incidence of organic disease, the mortality rate, or the recovery rate. On the other hand, left axial deviation is more common than a normal axis when other significant abnormalities are present in the electrocardiogram ; since the mortality rate is very much higher in the latter circumstances, it follows that

the prognosis is worse with left axial deviation than with a normal axis, when the remaining features of the electrocardiogram are not taken into account.

As was found to be the case with electrocardiograms showing a large Q3, the breadth of QRS is an extremely important prognostic feature in those with an " R′ " in lead III. The ultimate fate of 45 patients traced is compared in the table with the breadth of their QRS complexes. The mortality rate rises steadily and the recovery rate falls steadily as the breadth of QRS increases ; and there is such a close parallel between this series and the " large Q3 " series previously published that repetition of the latter figures seems justifiable for comparison.

Even when electrocardiograms which show other abnormalities are excluded, the breadth of QRS is still important for prognosis. This procedure considerably reduces the number with a broad QRS, as it cuts out all cases with bundle branch block or intraventricular block. We have traced 23 patients who had an " R′ " in lead III but no other significant abnormality. In seven, QRS was less than 0·08 sec. ; six are either improved or working and one is dead. In 16, QRS measured 0·08 or more ; four are dead and two are incapacitated, six have improved and four are working.

TABLE I

THE INFLUENCE OF THE BREADTH OF QRS

	CASES TRACED	DEAD, PER CENT.	INCAPACITATED, PER CENT.	IMPROVED OR WELL, PER CENT.
QRS below 0·08				
Large Q3	13	23	15	62
Present series	9	22	0	78 (44 working)
Total	22	22	9	68
QRS=0·08 sec.				
Large Q3	14	43	28	28
Present series	18	55	11	33 (11 working)
Total	32	50	18	31
QRS above 0·08				
Large Q3	15	73	13	13
Present series	18	72	6	22 (11 working)
Total	33	72	9	18

The Appearance or Disappearance of an " R′ " in Lead III

In four of our cases an " R′ " has appeared in lead III while the patient was under observation. They are as follows :

1. An obese woman of 36 with acute cholecystitis ; the development of " R′ " coincided with the onset of fatal circulatory failure immediately after operation.

2. A man of 30 with syphilitic aortitis, angina of effort, and gross RT depression in all leads of the electrocardiogram. "R′" appeared three weeks later, when RT had returned to the iso-electric level. A few hours after this record was obtained, he died suddenly. Post-mortem examination showed œdematous swelling of syphilitic plaques, producing almost complete occlusion of both coronary orifices, and the right circumflex branch contained a fresh thrombus; microscopic examination showed an interstitial myocarditis of older date.

3. A stout woman of 54 with hypertension and congestive failure had a falling blood pressure along with the electrocardiographic changes of coronary disease. At one stage there was an "R′" in lead III. Later the initial R disappeared, leaving S and "R′" only, accompanied by inversion of T1. She improved temporarily, but died from congestive failure three years later.

4. A man of 54 with hypertension developed auricular flutter during an attack of lobar pneumonia. "R′" appeared when the normal rhythm recurred spontaneously a few days after the crisis. Seven years later auricular fibrillation was present and "R′3" had disappeared; despite the fibrillation his capacity for effort was better than when he was convalescent from the pneumonia.

TABLE II

INCIDENCE OF HEART DISEASE EXPRESSED AS PERCENTAGES, AND FOLLOW-UP RESULTS

CLINICAL FINDINGS AND FOLLOW-UP RESULTS	1,200 CONSECUTIVE CASES FROM ORIGINAL MATERIAL.	352 CASES WITH NORMAL ELECTROCARDIOGRAM FROM SAME ORIGINAL MATERIAL	62 CASES WITH "SECOND POSITIVE WAVE OF QRS3" ("R′3")	36 CASES WITH "R′3" AS THE ONLY ABNORMALITY	26 CASES WITH SOME OTHER SIGNIFICANT ABNORMALITY IN ADDITION TO "R′3"	18 CASES WITH "R′3" AND PROMINENT INITIAL R (EXCEEDING 2 mm.)	7 CASES WITH "R′3," LARGE INITIAL R, AND NO OTHER ABNORMALITY	44 CASES WITH "R′3" AND SMALL INITIAL R (UNDER 2 mm.)	29 CASES WITH "R′3," SMALL INITIAL R, AND NO OTHER ABNORMALITY	14 CASES WITH QRS OF LESS THAN 0·08 SEC.	28 CASES WITH QRS OF 0·08 SEC.	20 CASES WITH QRS OF MORE THAN 0·08 SEC.
Organic disease :												
Rheumatic, per cent. ..	28	22	14	17	13	16	14	14	14	29	11	10
Syphilis and other infections, per cent. ..	5	1	3	0	8	0	0	4	0	0	7	0
Coronary disease Arteriosclerosis, Hypertension, per cent.	26	8	57	42	75	78	72	48	38	29	54	80
Pulmonary, Thyrotoxic, Others, per cent. ..	13	12	8	11	4	6	14	9	10	21	7	0
Total organic, per cent. ..	72	43	82	70	100	100	100	75	62	79	79	90
No organic lesion, per cent.	28	57	18	30	0	0	0	25	38	21	21	10
Cases traced.	—	—	45	23	22	16	6	29	17	9	18	18
Dead, per cent.	—	—	55	22	90	75	50	45	12	22	55	72
Incapacitated, per cent.	—	—	7	6	5	12½	17	3	0	0	11	5
Improved, per cent. ..	—	—	20	36	5	12½	33	24	41	33	23	11
Able for work, per cent.	—	—	18	36	0	0	0	28	47	44	11	11

In each of these four cases there are good grounds for associating the appearance of an " R′ " in lead III with the existence of an active pathological process affecting the myocardium.

In eight of our cases an " R′ " has disappeared at a later date. Its disappearance has only occurred while under observation in three cases. Two were cases of severe rheumatic carditis in which " R′ " was present during the acute stage and vanished as convalescence became established ; one of the patients made a good recovery, but the other was left with an adherent pericardium and died three years later. The third case was one of coronary disease with congestive failure, who became slowly worse and died two weeks after " R′ " had gone ; this is the only case in which its disappearance was not associated with at least temporary improvement. In the remaining five cases, the patient's capacity for effort was improved when on re-examination some years later " R′ " was no longer present.

Summary and Conclusions

1. Organic heart disease is more common in patients whose electrocardiograms show a " second positive wave of QRS3 " than in the material from which these cases were collected (82 against 72 per cent.). Many of these electrocardiograms show some other significant abnormality, in which case organic disease is always present, and the mortality since 1928–1933 has been 90 per cent.

2. Organic lesions are much more frequent in patients whose sole electrocardiographic abnormality consists of a " second positive wave of QRS3 " than in patients whose electrocardiograms are completely normal (70 per cent. against 43 per cent.). The increase is due to a greater number with coronary disease, hypertension, or arteriosclerosis (42 per cent. against 8 per cent.).

3. Where it has been possible to fix the time of appearance of the " second positive wave of QRS3," this has occurred when an active pathological process was affecting the myocardium. In seven out of eight cases in which it ultimately disappeared, the patient's capacity for effort improved, at least temporarily.

4. When a " second positive wave of QRS3 " is the only electrocardiographic abnormality, the shape of the complex is important for diagnosis and prognosis, and its breadth gives further assistance in prognosis.

5. As regards the shape of the complex, the height of the initial R is the determining factor. When this was a prominent deflection exceeding 2 mm. in height, organic disease was always present ; and the mortality in cases with an otherwise normal electrocardiogram was 50 per cent. When the initial R was a small deflection of less than 2 mm. in height the incidence of recognizable organic disease was only 62 per cent. and the mortality in cases with an otherwise normal electrocardiogram was only 12 per cent.

6. The breadth of QRS gives little help in diagnosis, but is of great importance in prognosis. The mortality rises steadily from 22 per cent. with a QRS below 0·08 sec., to 55 per cent. at 0·08 sec., and to 72 per cent. above 0·08 sec.

These mortality figures are almost identical with those found in dealing with another series of electrocardiograms where the abnormality was a "large Q3." The significance of the breadth of QRS therefore appears to be independent of the nature of any abnormality which may be present.

I am greatly indebted to Miss A. Miller, almoner at the Victoria Infirmary, for her valuable help in tracing patients ; to Drs. G. J. Wilson and A. E. S. Melville with the staff of the X-ray department ; to Dr. John Anderson, pathologist ; to the members of the medical and surgical staff ; and to the numerous practitioners who have kindly furnished information regarding their patients.

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SOME NOTES ON THE CARDIAC CLUB

BY

JOHN COWAN AND OTHERS

" The Sun of our body, the King and sole Commander of it." —
" Anatomy of Melancholy."

It is difficult to convey to the Georgian a proper impression of the attitude of the late Victorians to heart disease. Laennec's discovery of auscultation dominated the succeeding fifty years, and towards the end of the century the characteristic sounds of the various valvular lesions had been fully elucidated. For a time medicine stagnated. Those of us who were Residents in medical wards viewed the outlook with dismay, for the progress of surgery at that time, under the influence of Lister's work, was phenomenal. But better times were at hand.

The physiological laboratories were at work under Gaskell and others. The bacteriologists were turning their attention to medical subjects ; the tubercle bacillus was discovered by Koch. X-rays showed the contents of the thorax. The sphygmomanometer was adapted for clinical use, and the polygraph and later the electrocardiograph added their quota to the general store.

But, although new methods of investigation became available, a personal influence was needed to speed up the work, and James Mackenzie became the centre of the picture. He had many facets. A tall, burly man, with all the directness of the North, he seemed at first sight rather dictatorial and overbearing ; but his transparent goodwill, his unselfishness, and his humility soon made manifest that it was merely his intolerance of sham, and his search for truth. His intensive study of the Shorter Catechism had inculcated a downright mode of expression ! He took a kindly interest in all his neighbours, especially the younger members of his profession ; and he was always receptive of opinions based upon material which was outwith his own opportunities. Before long he had attracted to himself many men who were interested in the problems of heart disease ; and had himself suggested to some of our members that a small club should be formed for meetings and discussions. The Cardiac Club was conceived in Burnley.

Another European war came, and our profession was scattered over the world. It was not a great war, for it altered little save garden fences, but it left a terrible swathe behind it. Many men had died, and many more were suffering in health, from wounds or sickness, for whom provision was required. During the war the Pension Service was necessarily a makeshift ; the staff was insufficient and inexperienced, and their decisions needed revision and co-ordination. Many of the cases were cardiac, at least in name, so, in April 1920, the Ministry appointed Consultants to aid the Regional Directors in the assess-

ment and treatment of the cardiac patients. The majority of those appointed had been associated with James Mackenzie before the war.

In 1921 a conference of the Consultants was held in London, and others subsequently. They were mainly concerned with the technical and administrative difficulties of the Ministry, but several interesting discussions upon purely medical subjects took place, and some valuable statistical data were collected. The influence of war service as a cause of subacute bacterial endocarditis ; the effects of malaria upon the myocardium ; the incidence of syphilitic disease of the heart ; the causes of death in chronic valvular disease ; the value of quinidine in auricular fibrillation, etc., were considered.

The conferences were appreciated by those concerned (Carey Coombs, T. F. Cotton, John Cowan, Ivor Davies, Wardrop Griffith, John Hay, W. E. Hume, Thomas Lewis, J. E. MacIlwaine, W. T. Ritchie, Robert Sevestre, H. J. Starling), who felt that they had been of service to themselves and perhaps to cardiac science, and that it was desirable that they should be continued under different auspices. On February 21, 1922, Hume wrote to Cowan (apparently considering him to be the most senior of the group, though Wardrop Griffith was in reality the doyen) suggesting that the men interested in heart disease should be called together at the next meeting of the Association of Physicians. Accordingly a meeting took place at Oxford, with A. G. Gibson in the chair, and on April 22, 1922, the Cardiac Club was born. The official accoucheurs were Carey Coombs, T. F. Cotton, John Cowan and W. E. Hume. The first active meeting of the Club took place in London later that year. A list of the original members and of those elected subsequently follows, with the dates of election of the extra-ordinary members.

THE CARDIAC CLUB

Honorary Member

James Mackenzie, F.R.S., Kt., 1915

	Original Members	Extra-Ordinary Member
1922	Carey F. Coombs –1932	
	T. F. Cotton	
	John Cowan (*Secretary*, 1922–25)	1930
	J. G. Emanuel	1933
	A. G. Gibson (*Secretary*, 1925–28)	
	T. Wardrop Griffith, C.M.G.	1928
	John Hay	1935
	Thomas J. Horder, K.C.V.O., Kt. 1918, Bt. 1923, Baron 1933 .	1933
	W. E. Hume, C.M.G.	
	Thomas Lewis, C.B.E., F.R.S., Kt. 1921.	1927
	J. E. MacIlwaine, –1930	
	John Parkinson	
	W. T. Ritchie, O.B.E. (*Treasurer*, 1922–31)	1932
	H. J. Starling	
	K. D. Wilkinson, O.B.E.	

THE CARDIAC CLUB—*continued*

MEMBERS ELECTED SUBSEQUENTLY

1923 J. Crighton Bramwell (*Secretary*, 1928–32)
Francis R. Fraser
1924 A. N. Drury, F.R.S.
C. E. K. Herapath, M.C.
1925 A. M. Kennedy
S. Wentworth Patterson
1926 George A. Allan
Maurice A. Cassidy, C.B., K.C.V.O., Kt. 1934
1927 S. B. Boyd Campbell, M.C.
Arnold W. Stott
1928 H. Wallace Jones
D. Evan Bedford (*Secretary*, 1932–36)
1930 E. P. Poulton
1931 J. Maurice Campbell, O.B.E. (*Secretary*, 1936–37)
1933 R. T. Grant, F.R.S.
A. R. Gilchrist
1934 Leonard Abrahamson
C. W. Curtis Bain, M.C.
1936 C. Bruce Perry

Of these original members, three have died : Carey Coombs in his full vigour in 1932, MacIlwaine of Belfast in 1930, and James Mackenzie in 1925. At their next meeting the Club put on record this expression of appreciation of their only Honorary Member; and short notes of the dates and appointments of these three members follow.

Minute of the Cardiac Club, May 28, 1925

" The Cardiac Club laments the death, on January 26, 1925, of its Honorary Member, Sir James Mackenzie. By the power of his genius and the wealth of his achievements he exercised a vast influence upon the branch of Medicine with which his name is indissolubly associated ; and his pre-eminence, transcending the limitations of race and clime, was universally acknowledged. So long as the Science of Medicine endures so long will the name of Sir James Mackenzie be honoured.

" This tribute is rendered to the memory of him whom all Members of the Cardiac Club revered as their Master ; and the Club respectfully offers its sincere sympathy to Lady Mackenzie and Miss Mackenzie in the great loss which they have sustained."

JAMES MACKENZIE, 1853–1925

M.B., 1878 ; M.D., 1882 ; F.R.C.P., London, 1913. F.R.S., 1915. LL.D., Aberdeen, 1910 ; Edinburgh, 1911. Knight, 1915. Hon. Physician to H.M. the King in Scotland, 1920.

Physician, Victoria Hospital, Burnley ; 1880–1907. Physician, Mount Vernon Hospital for Diseases of the Chest ; 1910–18. Physician to the Cardiac Department, London Hospital, 1911–18. Director of the St. Andrew's Institute of Clinical Research, 1918–25. Schorstein Lecturer, 1911. Oliver Sharpey Lecturer, 1911. George A. Gibson Lecturer, 1914.

CAREY FRANKLIN COOMBS, 1879–1932

M.B., 1901. M.D., 1903. F.R.C.P., 1917.

Physician, Bristol General Hospital, 1920. Director, Bristol University Centre of Cardiac Research, 1927.

Major, R.A.M.C., T.F. Served in England, Egypt, Mesopotamia and France, 1914–19. Long Fox Memorial Lecturer, 1925. Chadwick Lecturer, 1927. Lumleian Lecturer, 1930.

JOHN ELDER MACILWAINE, 1874–1930

B.Sc. (Engineering) 1894. M.B., 1901 ; M.D., 1904 ; D.P.H., 1907.

Physician, Royal Victoria Hospital, Belfast, 1910–29. Professor of Materia Medica and Therapeutics, Queen's University, Belfast, 1921–28.

Irish Rugby International, 1897–99.

Dresser, Irish Hospital, South Africa, 1899–1900. Physician, St. John's Ambulance Brigade Hospital, France, 1915–17.

The First Annual Meeting of the Cardiac Club was held at University College Hospital, London, under the chairmanship of Sir Thomas Lewis on November 22, 1922 and, subsequent meetings have been held on the day previous to the annual meetings of the Association of Physicians, wherever the Association happened to meet.

Many subjects have been discussed by the Club: the heart in the acute infections ; hyperpiesis ; angina pectoris ; infarct of the heart ; heart block ; the heart in pregnancy ; cardiac asthma ; vaso-vagal attacks ; the treatment of œdema ; etc. In addition to the set subjects many short discussions upon case records, electrocardiograms, pathological specimens, etc., have also taken place, to the obvious pleasure of the members. On several occasions demonstrations have been given to the Club by those working in the laboratories. A list of the main subjects discussed at each meeting follows with the names of those who introduced the discussion.

In addition to the value the members have received from the meetings, the most important function of the Club has been the promotion of friendship between the members. All of us, from time to time, have received help from our confrères by advice or the supply of information or material. On two occasions the Club has been responsible for the mass collection of material from the members, from which important conclusions were drawn and made public at an earlier date than would have been possible if all the data had been dependent upon one observer. We refer to Hay's paper upon the value of quinidine in the treatment of auricular fibrillation and to Gibson's paper upon ischæmic necrosis of the heart. It seems desirable that this procedure should be repeated as occasion demands.

Webb's egg has grown and the stripling is thriving. Under the new constitution we anticipate a strong and healthy adult life.

JOHN COWAN AND OTHERS

GENERAL MEETINGS OF THE CARDIAC CLUB

1922.	April 22.	Oxford Decision to form the Cardiac Club	Chairman, A. G. Gibson
1922.	November 22.	University College Hospital, London First General Meeting	„ Sir Thomas Lewis
1923.	May 17.	Edinburgh	„ W. T. Ritchie
1924.	June 5.	Bristol	„ Carey Coombs
1925.	May 28	Royal Soc. of Medicine, London	„ T. F. Cotton
1926.	May 20.	Newcastle	„ W. E. Hume
1927.	June 2.	Belfast	„ J. E. MacIlwaine
1928.	May 24.	Liverpool	„ John Hay
1929.	April 4.	Cambridge	„ A. N. Drury
1930.	June 5.	The London Hospital, London	„ John Parkinson
1931.	May 21.	Manchester	„ Crighton Bramwell
1932.	May 12	Liverpool	„ John Hay
1933.	April 6.	Glasgow	„ G. A. Allan
1934.	May 7.	Leeds	„ Wardrop Griffith
1935.	June 6.	St. Thomas Hospital, London	„ Sir Maurice Cassidy
1936.	May 28.	Manchester Fifteenth and last General Meeting of the Cardiac Club.	„ Crighton Bramwell
1937.	January 8.	An Extra-Ordinary Meeting was held at Guy's Hospital, London (Chairman, Bramwell) at which it was decided that the Cardiac Club should be transformed into the Cardiac Society of Great Britain and Ireland.	

MAIN SUBJECTS OF DISCUSSION

1922.	Infective Endocarditis	Sir Thomas Horder
1923.	The Use of Quinidine	Hay and Parkinson
	Abnormal QRST Complexes	MacIlwaine
1924.	The Heart in Pneumonia	Wardrop Griffith
	Digitalis	Fraser and Andrus (introduced).
	The Effect of Adrenalin on the Heart	Hume and Fraser
	Heard Disease in Children	Cotton and Carey Coombs
1925.	Anæmic Necrosis of the Heart	Gibson
	Simple Tachycardia	Parkinson
	Blood Pressure	Bramwell and Carey Coombs
	Cardiovascular Syphilis	Cotton
1926.	The Heart in Hyperpiesis	Wilkinson
	Angina Pectoris	Starling
1927.	Heart Block	Kennedy
	Treatment of Œdema	Patterson
1928.	Angina Pectoris	Ritchie
	Injection of the Coronary Arteries	John Campbell (introduced)
1929.	Cardiac Asthma	Fraser and Emmanuel
	Vaso-vagal Attacks	Starling
1930.	Mitral Stenosis	Cowan
	The Heart and Athletics	Bramwell
1931.	Ætiology of Heart Disease	Carey Coombs
1932.	The Heart in Diphtheria	Hume and Wilkinson
	The Heart in Influenza	Herapath
	The Influence of Tobacco	Maurice Campbell
	The Influence of Alcohol	Patterson
1933.	Abnormalities of the T wave	Cowan and Allan
	Right and Left Ventricular Complexes	Drury
1934.	Diseases of the Pulmonary Artery	Parkinson and Bedford
	Vaso-motor Angina	Cassidy and Patterson
1935.	Heart Symptoms and Gall Bladder Disease	Cotton and Hume
	Cardiac Disease of Unknown Ætiology	Stott and Maurice Campbell
1936.	Heart Disease and Pregnancy	Bramwell
	Heart Block	Gilchrist

THE CARDIAC SOCIETY

After the meeting of the Cardiac Club in 1935 it was suggested that, with the increasing numbers who were interested in Cardiology, the time might have come for expanding into a larger and more representative society and that the Executive should consider this possibility.

At the 1936 meeting in Manchester a memorandum which had been drawn up by the Executive (Allan, Bedford, Drury and Wilkinson) provoked a long discussion. After general approval of the principle of expansion the following committee was appointed to draw up a scheme for submission to a special meeting of the club.

Members : Evan Bedford, Crighton Bramwell, F. R. Fraser, John Parkinson and Maurice Campbell (Secretary).

The committee held several meetings and drew up a draft constitution and rules for the Cardiac Society, and lists of names of those who should be invited to be Honorary, Extra-Ordinary, Ordinary and Associate members.

The last meeting of the Cardiac Club was a special one held in the Governor's Committee Room at Guy's Hospital on January 8, 1937. It was decided to accept these proposals for the formation of the new society, for its rules and constitution, and for the names of those who were invited to be the original members. The following were chosen as the first Council : Allan, Evan Bedford, Crighton Bramwell, Gilchrist (President 1937), Parkinson, Wilkinson and Maurice Campbell (Secretary and Treasurer).

At the First Annual Meeting of the Cardiac Society, held at Edinburgh on April 15, 1937, practically all the members attended, and in addition to the scientific business a committee was appointed to consider the possibility of starting a journal dealing with cardiology. *Members :* Gibson (Chairman), Evan Bedford, Maurice Campbell, Fraser and Wilkinson.

Several meetings were held, and in February 1938 the committee reported to the Council. They recommended that such a journal should be started and that the British Medical Association should be asked to publish it, as after various negotiations and meetings they thought that the British Medical Association were able and willing to publish a journal that would meet the needs of the Society. These recommendations were approved by the Council of the Cardiac Society, which appointed the Editors and the Editorial Board for the Journal. The title which has been chosen was suggested at an early stage, but many other titles have had their supporters. The action the Council had taken with reference to the Journal was approved by the Society at its Second Annual Meeting at Bristol on June 2, 1938.

MAURICE CAMPBELL,
Secretary of the Cardiac Society.

BRITISH HEART JOURNAL. EDITORIAL NOTE

For some time the need for a journal representative of British Cardiology has been apparent, and the Cardiac Society of Great Britain and Ireland, feeling assured of adequate support, has taken the initiative in starting the *British Heart Journal*. The Society's project has been facilitated by the generous co-operation oft he British Medical Association, which has undertaken the responsibility of publishing and distributing the Journal.

To those who hold that specialization has already gone too far, the appearance of another journal devoted to a special branch of medicine may seem a step in the wrong direction. In Harvey's time medicine had no offspring, for the surgeon was no more than the servant of the physician. One by one, Anatomy, Physiology and Pathology began to develop independently of the parent science, and in the course of time further separation has occurred, to meet the exigencies of practice, teaching, and research.

But even traditional divisions in medicine are not inviolate, and already the barrier between Medicine and Surgery is yielding, for example in the fields of Neurology and of Diseases of the Lungs. Radiology has become a special branch of medicine on grounds of expedience alone, for it is no more than a method of examination, and incidentally one which is readily applicable to the heart. Cardio-vascular radiology has been and is being developed as a means of diagnosis by those interested in the heart, whether primarily physicians, radiologists, anatomists or physiologists. The special journal represents an attempt at reorientation ; its proper function is to bridge the barriers separating existing divisions in medicine, and not to create new ones. By providing common ground on which physicians, anatomists, physiologists and pathologists may meet and pool their efforts in a particular direction, it enables those who treat the sick and those who work mainly in the laboratory to keep in contact with each others problems in the same field, to their mutual advantage.

The *British Heart Journal* will naturally reflect first and foremost the interests of members of the Cardiac Society, interests that are predominantly though not exclusively centred in the clinical aspects of cardio-vascular disease ; but the policy of the Journal, and indeed of the Society, will be to serve all those interested in the Heart and Circulation, irrespective of their calling.

In starting the *British Heart Journal*, we are not unmindful of its predecessor, *Heart*, founded by Sir Thomas Lewis nearly thirty years ago. During the period when knowledge of electrocardiography was developing, *Heart* rendered invaluable service to Cardiology and maintained a standard and reputation second to none among the scientific journals of the world. In changing the title to *Clinical Science*, Sir Thomas Lewis has indicated his intention of widening its scope and thus increasing its field of usefulness to those engaged in research. The Cardiac Society are indebted to him for his generous co-operation, and we take this opportunity of thanking him for his support and for contributing a foreword.

D. EVAN BEDFORD.